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## The Pathological Aspect of Phosphorus and Calcium Deficiency in Cattle.*

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At the Oxford Meeting of this Association in August, 1926, a paper was presented on "Minimum Mineral Requirements of Cattle" by Dr. P. J. du Toit, on behalf of the authors, Theiler, Green and du Toit, and subsequently published in full in *The Journal of Agricultural Science*, Vol. xvii, Part III, July, 1937. The material I wish to bring before you to-day represents a continuation of that work in its more pathological aspects, and to some extent completes the solution of a problem which has occupied my mind at various times over the last quarter of a century.

The starting point for our investigations was the disease lamsiekte, a disease of cattle in South Africa, known even amongst the earliest settlers as prevalent in certain areas, especially during dry seasons and after droughts. By the beginning of the present century it had become so serious as to render cattle-farming in many districts practically impossible. The cause remained a mystery until 1918, but was then cleared up by myself and co-workers on the basis of experiments carried out on the Government farm Armoedsvlakte in Bechuanaland. The results were published in our paper "Cause and Prevention of Lamsiekte," *Journal of the Department of Agriculture*, Union of South Africa, July, 1919, and I need only recall sufficient of that work for the purpose of the present paper.

The name "lamsiekte" signifies paralysis and indicates the principal symptoms. The disease proceeds without fever, pathological lesions are not very marked, and death may supervene in anything from a day to four weeks after the first clinical symptoms are noted.

In the course of our investigations it was observed that cattle displaying a depraved appetite, particularly for bones and other carcass debris occurring on the veld, were the principal sufferers. Since bone-chewing was the characteristic feature of the abnormal craving, we termed the symptom osteophagia. Examination of naturally-occurring veld debris revealed the presence of a toxin capable of producing the disease when administered *per os*, and of a toxicogenetic saprophyte subsequently identified as *B. botulinus* type D. The disease was thus established as parallel to the botulism known in human medicine, although, instead of occurring sporadically as it does in man, the bovine equivalent sometimes decimated the cattle population in certain areas. Prior to 1917 the mortality on our own experimental farm was 30 per cent. per annum, representing extinction of the herd in a few years.

Since our work has occasionally been misquoted in the literature, and the disease lamsiekte referred to as a mineral deficiency disease, it is perhaps necessary to emphasise again that lamsiekte is an intoxication and that the aspect of mineral deficiency is merely one link in the complete etiological chain. For the occurrence of the disease under natural conditions three factors must be present simultaneously: firstly, dead carcass material to provide the substrate for toxin production; secondly, *B. botulinus* type D to produce the toxin; and thirdly, osteophagia to induce the cattle to eat the toxic carrion which they would otherwise shun. It is with this last factor that mineral deficiency is concerned, and once we had reproduced the disease by dosing the toxin, determination of the precise cause of osteophagia became the central problem.

Why did herbivorous cattle turn carnivorous, and throw themselves like dogs on any old skeletons lying in the veld? It occurred to us that this abnormal behaviour might be a search for something lacking in the natural pasture and, since the soil of the farm (and of South Africa generally) was notoriously poor in phosphorus we turned our attention to this element. Animals showing osteophagia were dosed daily with liberal amounts of sterilised bone meal (three ounces to five ounces) and the abnormal craving rapidly disappeared. On substituting other phosphorus compounds, even pure phosphoric acid itself, for bone meal, the same results were obtained. No other substance produced the desired effect, and excess of calcium compounds even aggravated the craving. Analysis of the vegetation showed a marked deficiency of phosphorus (down to 0·07 per cent. $P_2O_5$ in the dry matter) but no noteworthy lack of other food essentials. The cause of osteophagia was thus pinned down to phosphorus deficiency and to nothing else. To clinch the matter, stabled cattle were fed upon hay cut from the area, and also fed, outside of the area, upon a synthetic ration (hay and maize endosperm) very low in phosphorus. In both cases osteophagia was produced, easily removable by supplementing the rations with bone meal or other phosphorus compounds.

In the course of the experiments the important observation was made that the naturally grazing cattle showed remarkable improvement in condition when supplied with bone meal; young stock grew more rapidly, and the milk yield of cows increased. It became quite apparent that osteophagia was merely the outward symptom of a deep-seated malnutrition, to which we applied the term "aphosphorosis" on account of its specific character. Furthermore, we observed that the minimum amount of phosphorus required to prevent osteophagia was not necessarily the optimum amount required for obtaining maximum growth and best condition. Hence aphosphorosis in its mildest form was displayed merely as "lack of thrift" and poor yield of beef or milk merely as "low production value." Phosphorus feeding then became highly important quite apart from the incidence of disease.

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In the course of the same series of experiments it was also observed that a degree of aphosphorosis greater than that required to produce mere osteophagia, was associated with a specific disease with a definite mortality incidence of its own, quite irrespective of any associated botulism. This disease is known in South Africa as "stijwesiekte," or "stiff-sickness." The name indicates the chief symptom, that is, stiffness of the gait, and the disease is most prevalent in young cows, particularly after calving, when the strain of milk production places an additional burden on the nutrition of the animal. In most cases the disease disappears after weaning, but may return on subsequent calving. In severe cases the animal may not recover at all. Affected cows lose condition, develop a stiff walk, frequently accompanied by lameness and thickening of the joints, particularly of the fetlock and the digits. Fractures of the bones may occur, and a cachectic condition may terminate fatally through inability of the animal to look after itself. In the veterinary literature this state of affairs is generally described as rickets and osteomalacia, although these terms are sometimes loosely applied without full knowledge of the etiology of the condition.

The question now arose in regard to our South African stiff-sickness: Is the pathological condition a pure aphosphorosis, or is it due to an association of several factors; does calcium also play a part as so commonly assumed in explaining the European osteomalacia? Is the deficiency of phosphorus associated with a deficiency of calcium? Or, finally, can the disease occur independently of mineral deficiencies, and in reality be only accidentally associated with them? To settle these points cattle were placed upon specially constructed rations, taking into consideration the modern views in regard to the causation of human rickets, the rôle of vitamins and the rôle of mineral balance. The results are described in the article already referred to. The series included rations low in calcium but rich in phosphorus, low in phosphorus but rich in calcium, low in both, and high in both. *A priori* we expected that if the disease appeared on the phosphorus-low diet, it would also appear on the calcium-low diet, since the two elements are used mainly for bone formation in the proportions required to deposit tri-calcium phosphate. Theoretically it should not matter much which element was made the limiting factor and we did not expect the surprising result actually obtained—a result which indicated that the calcium requirements of cattle are much lower than the phosphorus requirements.

The experiments were conducted on a physiological basis, and in order to secure the low mineral levels required for our purpose we had to construct semi-synthetic rations. The roughage was selected as poor in calcium and phosphorus and the quantity cut down to the minimum required to allow rumination to go on and avoid digestive disorder. Calories and protein were supplied chiefly as flaked maize endosperm, exceedingly low in all minerals. Although the hay-portion was limited to 3½ pounds per day, the maize endosperm was increased *ad libitum*, according to appetite. To this basal ration various mineral supplements were added, and in one case a vitamin supplement was included to serve as reference control (green forage). Our own earlier experiments, 17 years ago, had shown that vitamins A, B and C were not absolutely essential for cattle, or at any rate need not be included in the food; an observation which at that time (1914) we explained on the basis of possible vitamin synthesis in the alimentary tract of ruminants, but which we now leave open. We had, however, no qualms in designing a basal ration deficient in A, B and C. In regard to vitamin D, the intense solar radiation to which the cattle were exposed can be regarded as replacing the deficiency of D in the ration. The experiments were carried out at Onderstepoort, near Pretoria, at an altitude of about 4,000 feet and an average of seven hours a day brilliant sunshine. Hence, although the rations were not perfectly balanced from the point of view of the animal husbandryman, they were physiologically adequate when supplemented on the mineral side. The control cattle, receiving only bone meal in addition to the basal ration, developed normally and reached the same weight in the same time as the two reference controls receiving a vitamin supplement in addition.

The experiments were carried out on heifers and lasted from 18 to 27 months. At the beginning of the experiments the age of the animals ranged from nine to 18 months. The deficient feeding thus commenced considerably after weaning, but before the cattle were properly grown out. With one exception all heifers calved in due course, the period of pregnancy lying wholly within the period of experimental feeding. Calving, however, was in several instances accompanied by retention of afterbirth and subsequent metritic ailments.

Of the experiments discussed in the paper already mentioned, I have selected the following for pathological-anatomical consideration in the present paper:—

1. Heifers that received normal quantities of minerals, *i.e.* those that received a supplement of bone meal, bringing the basal ration up to about 37 gr. CaO and 28 gr. $P_2O_5$. These acted as controls and, despite the almost complete absence of vitamins A, B and C, remained clinically healthy, displayed normal growth and reacted the normal weight expected.

2. Heifers that received reduced quantities of bone meal calculated as below the borderline requirements of calcium and phosphorus. Actual intake per day amounted to about 23 gr. CaO and 17 gr. of $P_2O_5$. These slowly developed symptoms of stiff-sickness, which became pronounced after calving.

3. Heifers that received a ration cut down below 6 gr. $P_2O_5$, but left adequate in calcium, 29 gr. CaO by addition of chalk. These showed symptoms as grave as those deprived of both calcium and phosphorus. Indeed, one of the two animals showed even more marked clinical stiff-sickness than those exposed to the double deficiency.

4. Heifers that received phosphorus 24 gr. $P_2O_5$ in the form of a bran supplement, which left the ration still notably deficient in calcium, about 9 gr. CaO, as judged by conventional standards. Despite the low calcium, *a priori* designed to produce a calcium deficiency disease, the animals remained healthy, developed normally, and showed no clinical symptoms whatever.

5. Heifers on the basal ration deficient in both phosphorus and calcium; phosphorus down to less than 6 gr. $P_2O_5$ per day and calcium about 7 gr. CaO. These developed disease, although no more acutely than those subjected to phosphorus deficiency alone.

From the results of these experiments we were satisfied :—

(*a*) That we had produced, by artificial feeding, a type of mineral deficiency disease, simulating the naturally occurring stiff-sickness in its severest form.

(*b*) That phosphorus deficiency plays the dominant rôle; and that since the daily intake of calcium of animals grazing over our known stiff-sickness areas was much higher than the calcium intake of our experimental animals, the naturally occurring disease was a simple aphosphorosis. This conclusion was abundantly confirmed by field experiments in the stiff-sickness area (Ermelo district), in which we showed that the natural disease could be prevented and cured not only by bone meal, supplying calcium and phosphorus, but equally well by bran, supplying abundant phosphorus with hardly any additional calcium. Indeed, although the pasture was low in calcium, about 0.3 per cent. CaO, a supplement of calcium (chalk) without phosphorus actually aggravated the incidence of stiff-sickness. Apparently the low calcium of the pasture was more than sufficient to balance out the still lower phosphorus, so that the latter was the limiting factor.

Of additional interest in these experiments was the fact that the inorganic phosphorus of the blood of the animals on the phosphorus deficient rations dropped very markedly long before clinical symptoms appeared, so that thereafter we could use a determination of blood phosphorus in the grazing cattle as a guide to the phosphorus deficiency of pastures, and as an indication of aphosphorosis in the animals themselves.

The next question to be settled was the identity of the disease in relation to the already known diseases of animals. Since stiff-sickness is an aphosphorosis, pure and simple, with what known disease of the skeleton can it be identified? I propose to answer that question to-day, on the basis of my microscopical study of the bones.

The clinical picture corresponded to rickets and osteomalacia and I can now say with certainty that the pathological-anatomical diagnosis is unquestionably the same, provided we adhere closely to the correct definition of the diseases. In human pathology an exact definition has been given to the terms "rickets" and "osteomalacia," based primarily on the nature of the bone lesions, and in our interpretation of changes in animals supposed to be suffering from similar diseases we must be careful to adopt the same primary conceptions.

There is a general consensus of opinion that between the two diseases osteomalacia and rickets there exists no fundamental difference. The distinction is simply one of age, depending upon the stage of skeletal development, and osteomalacia of adults is regarded as corresponding to rickets of adolescents. The pathognomonic change upon which the diagnosis is based is the presence of osteoid tissue in quantities surpassing normal physiological limits. In young growing animals the alterations in the epiphyseal cartilages have attracted most attention, and the term "rachitic" applies more specifically to them. But the osteoid substance may be found in pathological abundance everywhere in the skeleton, in all bones of both adolescent and adult, and the more universal changes are termed osteomalacic. After closure of the epiphyses we may have osteomalacia without rickets. The changes found in our heifers, and which were produced by simple phosphorus deficiency, are unquestionably those of rickets. The animals were still growing and the epiphyseal lines were not yet all closed by ossification. Typical rachitic lesions, corresponding to the ages of the animals, were found in the epiphyses, while in the remainder of the skeleton the changes generally called osteomalacic were present in varying degree. At the same time the osseous tissue showed advanced atrophy, and this atrophy appears to be the primary alteration which precedes the formation of osteoid. In my opinion the osteoid tissue in these heifers appeared only when the organism was deprived of the minerals necessary for normal ossification, *i.e.*, when the phosphorus and calcium lost to the body by the normal processes of physiological elimination were no longer balanced by a corresponding replacement from minerals in the food. The animals must have passed into a state of negative calcium and phosphorus balance, and excessive formation of uncalcified osteoid tissue appears. The very marked atrophy found in the heifers perhaps represents a point of difference from human rickets, but since a certain measure of atrophy may also be found in the human disease, the distinction is not fundamental. I regard it simply as one of degree.

The question has now to be answered as to whether we are justified in claiming a complete identity between human rickets and our straight aphosphorosis of young cattle. If we confine ourselves to the pathological lesions the answer is in the affirmative, but if we consider the question from the etiological point of view the answer is not so simple. In the human subject the disease may be observed even when the diet contains quantities of minerals proportionately higher than did the diet of our heifers. The determining factor is, then, the absence of vitamin D, or its hygienic equivalent of ultra-violet radiation. In our experiments the D factor was amply supplied by the intense insolation of a sub-tropical sun, with only the natural hair covering as protection for the cattle. The only missing factor of any importance was the phosphorus, but in human medicine this never seems to have been recorded as a limiting factor causing rickets or osteomalacia. Both of these diseases seem to be practically unknown in the human subject in South Africa and amongst animals it is only the herbivora on certain defined pastures which suffer from them: never the omnivora, which receive a proportion of phosphorus-rich cereals in their diet. Why this difference? In claiming identity between bovine stiff-sickness and human rickets and osteomalacia, I explain matters as follows: Vitamin D, or its equivalent ultra-violet ray, is generally accepted as regulator for the fixation of calcium and phosphorus in osseous tissue. When this is deficient, as it is generally the case in the circumstances under which human rickets occurs, there may be defective ossification even in the presence of adequate phosphorus and calcium in the food. In the case of our heifers the D factor was abundant to fix the phosphorus and calcium, but the phosphorus itself was not there to be fixed. The newly-forming tissue therefore developed as uncalcified osteoid. Apparently it comes to the same thing, so far as the bone is concerned, whether the minerals are there but cannot be utilised or whether they are not there at all. The ossification system seems to demand the presence of Ca, P, and D, and the absence of any one may be expected to induce the same end-result, *i.e.*, the laying down of osteoid instead of true bone—indeed, often the

laying down of abnormally large quantities of such uncalcified material in the attempt to compensate in mass for what is lacking in strength.

Looked at in this way, the etiological difference between rickets and osteomalacia in the human subject and stiff-sickness in the bovine does not seem so anomalous, so that we need not hesitate to regard advanced bovine aphosphorosis and human D-avitaminosis as identical pathological conditions. The cause is different but the effect the same.

Now in regard to experimental investigation of rickets in Europe and America, under conditions of mixed deficiency of minerals and vitamins, the white rat has played an important rôle as laboratory animal, and the rachitis of the rat, produced by feeding diets deficient in minerals, with or without the D factor, has been regarded as identical with human rickets. The lesions have been sub-classified in various ways, but in regard to causation the emphasis is frequently laid upon disproportion in the relative amounts of calcium and phosphorus rather than upon simple deficiency of either. In some cases an excess of phosphorus rather than a deficiency of calcium, or an excess of calcium rather than a deficiency of phosphorus, has been incriminated, and attempts have been made to lay down an optimal ratio of Ca : P in presence of vitamin D.

This stress on ratios is not in accordance with our experience in cattle, and within fairly wide limits we consider the absolute intake of the mineral present in least amount as the determining factor. We regard the problem from the point of view of the " limiting factor " without, of course, denying that optimal ratios play a subsidiary rôle. Since so many investigators (notably Marek) stress the question of " mineral balance " or ratios, it is perhaps not out of place to insist that this is subsidiary. The normal physiological mechanisms for maintaining a constant level in the blood which nourishes the tissues outweigh wide differences in the rations. If, of course, there is such a preponderance of Ca over P in the food that it depresses the solubility of the digested phosphorus compounds in the gut, then we would expect an aggravation of phosphorus deficiency in diets already very low in that element. But the importance of the disproportion only affects rations on the borderline of phosphorus deficiency, and should be treated as distinct from ratio which may vary quite widely without detriment to the animal so long as the " limiting element " is well above the minimal quantity.

In the case of our heifers on minimal $P_2O_5$ with about six times as much CaO, the animals developed symptoms faster than they would have done with the same phosphorus and less calcium. But the primary phosphorus deficiency here was very acute, and if both phosphorus and calcium had been increased five-fold, so securing adequate phosphorus but the same excessive ratio of calcium, there is no doubt that rickets would not have developed at all. Furthermore, from the practical grazing point of view, there are thousands of acres of pasture in South Africa in which the calcium dominates very markedly over the phosphorus without producing disease. Disease only appears when the absolute amount of phosphorus in the pasture drops below the minimum physiological requirements of the grazing animal. Roughly speaking, we expect aphosphorosis in a mild form (osteophagia) when the $P_2O_5$, expressed on the dry matter of the winter grass, falls below 0·2 per cent., and in acute form (rickets and osteomalacia) when the phosphorus drops to about 0·1 per cent. or less on the dry matter of the mature grass. But whether the calcium is 0·6 per cent. CaO, as it may be around Vrijburg, or only half this, as it may be around Ermelo, does not seem to matter very much. These figures are, of course, only illustrative. To express them more accurately they would need to be stated in relation to the energy value of the grazing, *i.e.*, as so much phosphorus per therm (thousand calories).

As a further deduction from our experiments on heifers we can also say with tolerable certainty that a straight calcium deficiency disease, *i.e.*, a simple acalcicosis, is never likely to occur in cattle under natural conditions. That is to say, it is not likely that any natural pasture, adequate in all other nutritional respects, will ever be found with a calcium content which provides a daily intake of CaO below that of the synthetic ration upon which we failed to produce a calcium deficiency disease in our experiments.

What the " limiting intake " of calcium for cattle actually is we cannot yet say with precision. In designing the experiments we thought that with the heifers a basal ration with bran supplement, the quantity of CaO (about 9 gr. per day) would be below the physiological minimum and that an " acalcicosis " would be produced, similar perhaps to the " aphosphorosis " produced on the phosphorus low rations. But apparently even this small quantity of calcium sufficed for normal healthy development and we concluded that the requirements of cattle for phosphorus are much higher than for calcium. Microscopical examination of the bones of one of these heifers revealed slight osteoporosis but no sign of rickets or osteomalacia. It seems probable that if the calcium had been reduced still further, say 6 gr. CaO per day instead of 9 gr., the deposition of tricalcium phosphate in the osseous tissue would have been hampered and excessive quantities of osteoid have been formed, so producing a form of osteomalacia and rickets due to acalcicosis. But that experiment has yet to be performed, and it is not inconceivable that the picture would then be complicated by other manifestations of calcium deficiency affecting other tissues besides the bones (glands). I do not think, however, that any natural pasture exists capable of supplying adequate calories, protein and other minerals and yet so low in calcium that the daily intake would supply so small a quantity of CaO as 6 gr. Although in stiff-sickness we have a widespread naturally occurring aphosphorosis, it is not probable that we will ever encounter an acalcicosis in practice. In the mature leafy part of the plant Ca dominates over P, just as in the seeds P dominates over Ca.

Finally, I would like to add that in comparing our acute bovine aphosphorosis with human rickets and osteomalacia I do not deny the possibility that bovine rickets could occur under conditions which produce the human disease, *i.e.*, in presence of sufficient phosphorus but deficiency of vitamin D. Such conditions, however, could not occur under practical pastoral conditions. If they occur at all it would be under artificial conditions of unhygienic stall feeding. From the practical point of view rickets and

osteomalacia in cattle that have the usual access to pasture, should therefore be viewed chiefly from the point of phosphorus deficiency in the first instance. And then these diseases should only be regarded as the definite advanced stages of an aphosphorosis which, in milder clinically unrecognisable form, may cause serious production losses in beef, milk, and calf crop. Osteophagia, probably associated with a certain degree of osteoporosis, is a premonitory symptom.

The investigations of which I have given this short review have had far-reaching economic consequences in South Africa. Once it was recognised that osteophagia was a symptom of aphosphorosis, stiff-sickness a definite advanced stage of the same disease, and lamsiekte an accidental intoxication due to botolinus infection of skeletal débris, but thus indirectly connected with osteophagia, and the prevention of the latter disease the cure of the former, then the general improvement in production became possible through the simple administration of phosphates in the form of bone meal.

In Bechuanaland, where our original experiments were carried out, phosphorus deficiency rendered cattle farming a precarious venture. To-day, I am informed, thanks to these investigations, the same area carries a flourishing dairy industry, and "Armoedsvlakte," the name of our experimental farm, is a word to conjure with amongst the local community.

# Pregnancy Disease in Ewes.

## A—In South Africa.

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Under the titles "Pregnancy Disease of Sheep," "Acidosis," "Fatty Infiltration of the Liver," "Pregnancy Toxæmia," "Acute Parenchymatous Hepatitis," and "Domsiekte," a condition affecting pregnant ewes during the last stages of gestation has been described in countries as far apart as America, England, New Zealand, Hungary and South Africa.

There is as yet no justification for stating definitely that the condition is one and the same in all the countries mentioned, though there is such a close resemblance as regards the incidence, symptoms and *post-mortem* appearances that one may reasonably expect the various conditions mentioned to be identical.

The disease has been described at length by various observers, and my only reason for again drawing attention to it is to acquaint my colleagues in various parts of the world with the success which has attended our efforts to find a suitable remedy in South Africa.

### Occurrence.

There is no record of the disease having been prevalent in this country prior to 1924. If it did occur previous to that date, it was only to a limited extent which did not merit serious consideration. But in subsequent years it spread rapidly, and in 1926-27 assumed alarming proportions among flocks in the Karroo portion of the Cape Province and in the Southern Free State, some farmers losing as many as 300 ewes. These parts form some of the driest portions of the Union of South Africa, and sheep there live almost exclusively on the Karroo bush. This, however, provides excellent grazing and normally the parts referred to are beyond doubt the premier merino sheep districts of the Union.

During the last decade the areas concerned have been subjected to an unprecedented series of droughts, while at the same time there has been a tendency on the part of farmers to overstock their farms. The inevitable result is that the vegetation has become greatly impoverished, and it is considered that the appearance and severity of the disease are related to these two factors.

### Symptoms.

The disease has only been noticed in pregnant ewes during the last two or three weeks before lambing. A few exceptional cases occurring within two or three hours after lambing have also been reported. Old ewes and those bearing twin lambs seem to be more susceptible. Condition does not appear to play an important part, but if anything ewes in good condition are more susceptible. This appears to contradict the statement that the prevalence of the disease is associated with drought and overstocking, but it should be borne in mind that an animal in good condition is not necessarily well nourished.

Affected ewes lag behind the flock and stop feeding. They appear dull and dejected and may stand in the same spot for hours. In some cases there is blindness and frequently nervous symptoms are shown, such as grinding of the teeth, walking in circles or star-gazing. The name "Domsiekte" given to the disease by South African farmers is very appropriate, "dom" being the Dutch for stupid or dopey. Rapidly progressive weakness is shown, the ewe goes down and is unable to rise again without assistance. She then lapses into a semi-comatosed condition, and may remain like this for hours and even days before death supervenes. If the ewes are killed when in a condition of coma and the abdominal cavity immediately opened, the fœtus can in most cases be extracted alive.

### Post-Mortem.

There are no marked characteristic lesions to be found on *post-mortem* examination, with the exception of fatty infiltration of the liver. This condition may also sometimes be seen in normal pregnant ewes and it is impossible at this stage to state its relationship to the actual disease. There is evidence of sluggish peristaltic movements as shown by the quantity of fæces often found in the intestines. The fat of the abdominal cavity sometimes has a mottled appearance.

### Treatment.

During the 1926-27 outbreaks of the disease it was noticed that no cases occurred among flocks that were habitually supplied with a bonemeal lick. It was accordingly decided to recommend owners of flocks in which the disease was prevalent to feed pregnant ewes sterilised bonemeal. In bad outbreaks they were advised to dose all ewes, giving each one ounce of bonemeal daily, and further to supply a lick consisting of three parts bonemeal and one part salt.

The results surpassed our most sanguine expectations. Without exception the disease was checked within a few days. As farmers in the affected areas have consequently

come to look upon bonemeal feeding as indispensable, this is now being carried out as a matter of routine. The result is that domsiekte disappeared in 1927 and has not been heard of since.

Sterilised bonemeal was also used with good results as a cure. Ewes in an advanced stage of the disease were dosed with a tablespoonful three times daily. Recovery followed in the majority of cases within three or four days, and such ewes then gave birth to normal, healthy lambs.

### Etiology.

The incidence of the disease and the success of bonemeal treatment suggest that in South Africa at any rate nutrition plays an important part, and moreover that phosphate and calcium are intimately concerned in the causation of the disease. On account of the deficiency of phosphate in South African soil and vegetation it was at first considered that domsiekte may be due to a phosphate deficiency. This disease, however, bears no resemblance to the well-known aphosphorosis of cattle.

To those observers who have seen pregnancy disease in ewes the similarity of its symptoms to those of milk fever must be apparent, the only notable difference being that it does not run such a rapid course.

Owing to the effective method of prevention, material for further investigation is unobtainable, and there is no record of the calcium content of the blood of affected ewes having been determined in South Africa. Nevertheless, one feels justified in expressing the opinion that pregnancy disease may be a hypocalcæmia. The difference between it and milk fever would be this, that whereas in milk fever there is a sudden drain on the blood calcium at the time of calving in order to form colostrum, in pregnancy disease the calcium would not be required so much for the colostrum and milk as for the fœtus. It is probable that during the last two or three weeks of pregnancy there is a heavy demand on the blood of the ewe for calcium to satisfy the needs of the rapidly growing fœtus, and where the intake is not sufficient to meet this demand a hypocalcæmia results, this giving rise to the symptoms of pregnancy disease.

The fact that ewes bearing twin lambs are more susceptible to the disease lends support to this theory, which also explains why inflation of the udder does not have the same beneficial effects as in milk fever.

It is trusted that members of the profession in other parts of the world will try out the treatment that we have found so successful in South Africa. The results obtained by them should show to a large extent whether the disease is the same in all countries. Further, the facts given should indicate to investigators able to obtain material lines on which further investigations can be pursued, and thus enable them to determine definitely the cause of the disease.

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## B.—In New Zealand.

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During the lambing seasons of 1930 and 1931 I have been interested in the factors of predisposition and causation of pregnancy disease in ewes. In New Zealand the losses from this disease are enormous and I consider that the recommendations in regard to exercise and excessive fatness of condition are not free from blame.

Until June, 1930, nothing of any moment was known in regard to prevention except exercise and barer feed. For two years now I have been investigating this disease and the conclusions I have arrived at concerning the chief factors of predisposition, apart from the presence of big lambs *in utero,* are (1) underfeeding and (2) unbalanced feeding. I consider that exercise has nothing to do with the prevention of the disease and in this I strongly disagree with all other writers on this disease. In my view the results attributed to exercise in the past have been interpreted without reference to the regulation of the food supply and therefore results attributed to exercise alone must be likened to ghosts which are believed to exist but never have been seen.

The views of Hopkirk in *The New Zealand J. Agric.* are in the main the same as I supplied to him in a private communication in September, 1930. That the best ewes on the best pasture are affected is not the case as far as my observations go. It would be of interest to know what Hopkirk means by "best" as applied to pastures.

Bosworth and Glover, in their paper to the N.V.M.A. Conference, overlook the fact that the kidneys are not infrequently affected with fatty changes. I agree with the latter writer that glucose is of value. About forty per cent. of cases treated by me responded to the oral administration of treacle (1½-lbs. for big ewes, ¾ to 1-lb. for medium ewes, and ½ to ¾-lb. for small and weak ewes) and the hypodermic injection (sometimes several injections) of an isotonic solution of glucose (dose usually 16 ounces). To obtain cases in the early stages (as treatment, in my experience with about two hundred cases, is practically hopeless in old-standing cases) ewes in affected flocks were submitted to forced exercise. This brings on the prodromal symptoms of impaired gait, etc. Excitement appears to be the earliest symptom.

In New Zealand it is a common practice for sheep farmers to take their ewes for a walk along the road during the last three or four weeks of the gestation period. The very beneficial effects so often claimed are, in my opinion, due to the feed that is picked up. This year I have been able to demonstrate clearly that exercise should only be considered in so far as it is embodied in good ewe husbandry. Ewes carried at the rate of some twenty-four to the acre, receiving no exercise, did not develop the trouble. Weights of lambs at birth permit the statement that susceptible animals were present in all lots under experiment.

I have failed so far to diagnose a case of pregnancy disease that was due to excessive fatness of condition. The general condition of the flock is not a sure guide to the

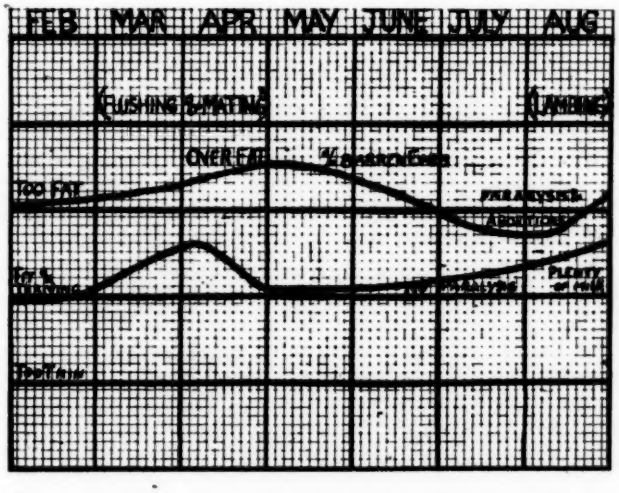
condition of the one or two cases that may develop the disease. This is particularly true in the case of well fed sheep. It is common knowledge that all ewes do not lend themselves to hand feeding. Observations and experiments conducted here demonstrate that the weights of ewes during the gestation period and the presence of big lambs *in utero* and whether or not susceptible animals eat, if hand fed, or receive properly balanced feed if on natural herbage, decide whether ewes will develop the disease.

The disease has been produced experimentally at the College. It has also been demonstrated here that the condition does not develop suddenly but results from the cumulative effects of underfeeding or unbalanced feeding.

Loss of weight occurred in all experimental animals that developed *ante-partum* paralysis here. Weights and the chemical composition of the feeds allowed the experimental animals showed clearly either underfeeding or unbalanced feeding.

A high incidence of dead or weak lambs at birth or dead lambs *in utero* and abortions is a not uncommon accompaniment to a high incidence of pregnancy disease. They appear to be allied conditions of nutritional origin.

Factors such as flushing, age, weight of ewes during the gestation period, weight of lambs at birth, quantities of feeds used, etc., have been investigated here. Bulletin No. 22, prepared by this college, a copy of which I enclose, illustrates by means of a graph the regulation of the food supply of in-lamb ewes as advocated by us and found successful in New Zealand.




"The graph shows that, disregarding the irregularity at flushing time, there is, in the case of healthy ewes, a gradual improvement in condition until lambing is complete. If, however, ewes are over-fat at mating time, the accomplishment of this procedure becomes very difficult when consideration is given to weather conditions and the normally reduced winter food supply. Under natural conditions of flock management the graph line will alter according to the available food, as influenced by weather conditions. Under intensive sheep farming conditions any marked deviation in the graph line can generally be minimised by modifying the food supply and shelter."

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The unveiling at Brussels of a war memorial to the carrier pigeon has caused bitter comment as to why Great Britain has shown no disposition to pay a similar tribute to the 250,000 horses killed or wounded on the western front.

# Acetonæmia.*

R. Hudson, F.R.C.V.S., Retford.

Definition.

The presence of acetone in the blood does not constitute a disease but only a symptom. What the disease is and what is its cause are still debatable points.

It appears to be a toxæmia characterised by the smell of acetone in the secretions and great wasting.

History.

I believe that it has been known for several generations as "second cleansing" and for quite a long time as "post-partum dyspepsia."

Etiology.

(1) Hutyra and Marek state "The etiology is open to conjecture."

(2) The condition is variously described as being due to—

(*a*) Absorption of aseptic lochia;

(*b*) Toxins formed in the udder;

(*c*) Too rich and unaccustomed foods;

(*d*) Processes of decomposition in stomach and intestines, with abnormal catabolism of protein.

(*e*) Increased fat and protein catabolism with increase of acetone compounds (beta-oxybutyric acid, diacetic acid and acetone) and transmigration of fat to liver for conversion into sugar. (Hence the liver enlargement and infiltration). Acetone compounds follow insufficient combustion of carbo-hydrates and this points to deficiency of adrenalin, following the exhaustion of parturition and milk secretion and possibly increased tonus of the vagus and insufficient conversion of glycogen to sugar following parturition and milk production.

(*f*) Acidosis, due to increased production of diacetic and beta-oxybutyric acid with neutralisation of alkalies of the blood, thus bringing about the presence of stupefying carbonic acid and other waste products of metabolism.

(*g*) Hypo-glycæmia. It has been found by Greig and Dryerre that there is a deficiency of glucose in the blood.

I am not sufficient of a biochemist to deal with the complex theories involved in the production, conversion and balancing of food constituents in the blood nor their bye-products, but one can easily see that as the result of various conditions and diseases they may be easily upset. After I became familiar with the smell associated with the disease I often wondered in what disease connection it seemed so familiar to me and one day it occurred to me that I had often noticed it in my children when they had suffered from their various ailments. Later I found this infantile condition is an acidosis (with acetone, etc.) associated with metabolic disturbance following gastro-intestinal symptoms and fever and that fats should be avoided in the diet; also that it soon disappeared if soda-bicarbonate and glucose were administered.

(*h*) The possibility that the disease is due to some bacterial changes taking place in the contents of the uterus, following the early closing of the os, has always been present in my mind. At one time I was prepared to doubt it owing to the fact that I recognised the presence of

*Paper presented to the Lincolnshire and District Division, N.V.M.A., at Lincoln, on October 8th, 1931.

acetone in the breath before calving in a few cases, but what has held me to the theory has been the number of cases which have yielded to uterine treatment.

Occurrence.

The disease is most frequent in milch cows after the third calving and increases up to about the sixth calf —though heifers may suffer. It is most common in small herds. It is mostly met with during the early spring and after close housing. For a long time I never saw a case during the summer: this summer, however, has proved an exception, for I have had two.

Various writers have placed its appearance at 8 to 17 days after calving, others at two days to several weeks or even several months.

The occurrence of acetonæmia is said to be influenced by a fall in the barometer. It is said to be rare in the south of England and to increase as one progresses north.

I have often noticed the acetone smell during the course of milk fever attacks, but as in those instances in which it is present before calving, it vanishes with the recovery of the animal. I remember one case of milk fever where the smell persisted for a fortnight, but this case never yielded to milk fever treatment and eventually died.

Symptoms.

We may have mild, severe and nervous types of the disease. Some cases pass quite unnoticed by the owner but may be recognised by the veterinary surgeon familiar with the smell of acetone. Sometimes I have found it difficult to convince the owner that there was something abnormal.

There are gradual failing of the appetite, constipation, lasting depression, reeling gait, inco-ordination of movement, slowing of respiration and pulse rate. Severe cases show increased heart action, increase in size of liver and sensitiveness to pressure over it. (I have not noticed this.) The smell of acetone is present in breath, milk and urine; urine reaction, acid—specific gravity falls to 1005 and below (lowest normal S.G. 1014).

Some cases show nervous symptoms—blindness, string-halt movements in the limbs, epileptiform attacks, pushing against the wall, champing the jaws, biting the manger, racks, and even their own limbs.

If they fall they may be unable to rise and they lie kicking, as in a fit of epilepsy.

Diagnosis.

The association of the acetone smell with a cow recently calved may be sufficient, but diagnosis may be aided by the application of Rothera's test for acetone in the urine.

Treatment.

It is advisable to place the animal in a good loose-box, well ventilated and well bedded.

Open-air treatment should be sought after if possible and the animal rugged down and placed in a safe paddock —I say "safe" because, if allowed to wander, she may fall into ponds or dykes and need pulling out.

Subjects of this disease are often difficult to feed and I do not know anything which they prefer—one has to keep trying them with bits of various foods and the less rich they are in proteins and fats or oils the better. I often find they will take sliced potatoes in meal, oatmeal, barley meal or bran as well as anything.

Treacle is useful—two or three pounds daily in gruel as drink or administered.

An effort should be made to increase the combustion of carbohydrates and so check the production of acetone, by feeding easily digested foods rich in carbohydrates, such as boiled potatoes, syrup, molasses, etc. (Hutyra and Marek).

Various drugs have been suggested, *viz.*, formalin (Imrie), soda bicarb. up to 12 ounces daily; aloes 1 ounce daily; ammon. carb., nucis. vom., soda bicarb.; mag. sulph., gentian, chloral hydrate, in small doses; pot. nitrate and glucose.

It would take too long to deal with treatment in detail, but I would like to remark that a Yorkshire veterinary surgeon (I am sorry I have forgotten his name for the moment) wrote me that he had found chloral hydrate in small doses a specific for the disease, but I have been so busy trying other things that I have not had a chance to test it.

Pot. nit., with tonics, is useful in getting rid of acetone by way of the kidneys and bowels.

**Subcutaneous Injection of Glucose.**

Since the use of a large dose of glucose (9 ounces in 3 pints of water, injected subcutaneously) was suggested by Greig and Dryerre and reported on by Stinson, I have made several trials of it and will quote you cases for and against the treatment.

No. 1.—This was a second-calf heifer, calved one month, ailing a week. Acetone smell bad in breath and milk. Injected nine ounces glucose in 24 ounces water (wine bottleful in all), half each side of neck.

Second day—no improvement.

Third day—smell slight in breath, not in milk; feeding and looks much better; milk increasing.

Seat of injection on right side a little thickened and tender.

No. 2.—A fourth-calf cow, feeding and milking well, but shrinking.

She had two injections of glucose at a few days' interval; showed temporary improvement and relapsed.

Along with the third injection her uterus was injected with a weak creolin solution and on the following visit she showed considerable improvement.

This cow was badly fed during treatment and did not get sufficient food to supply her milk yield.

No. 3.—A first-calf heifer. She showed only temporary benefit from injections and some weeks after, though smell had vanished, she was feeding badly. I think she has some urinary trouble, for she goes about with her tail raised.

No. 4.—This cow had a very strong smell and had been feeding badly for a fortnight.

She had ammon. carb., soda and nux powders for two or three days and showed some improvement.

April 3rd.—She received 9 oz. glucose injection.

April 6th.—The smell was much less noticeable and she was feeding better. The day following the injection she was depressed and took little food. The skin was slightly thickened at the area of injection. Later she did not pick up as fast as I thought she would and I intended to inject her again but, when visiting, she was improving so I decided to leave her alone.

No. 5.—Had been ailing slightly for some time. Not injected. Was given ammon. carb., soda and nux. powders

and two days later was feeding better and smelling less.

She was going out, but went off her feed again—probably a chill, as the weather became bad. There was now no smell. Later—feeding better, but carries her tail up a little.

No. 6.—Made rapid improvement with ammon. carb., nux vom. and soda powders. Not injected. Turned out to grass. Four days later she had lost the acetone smell and was feeding and milking well.

No. 7.—Cow noticed with smell in breath and milk; scouring; Johne reactor. She was feeding fairly well and milking heavily. The owner had not noticed anything amiss, but I observed her when attending another cow.

Two days later she had a seizure and fell, struggling. On visiting, I found her up, biting the manger and propping herself on the side of the stall. I injected nine ounces of glucose in 48 ounces water. She recovered from the fit in two to three hours.

Next day the cow was much better. An injection of half the quantity of glucose was repeated daily for three days and she recovered. Eventually, however, she went off with Johne's disease.

No. 8.—An old cow, a heavy milker, which had milk fever: her temperature was 105° F. and there were signs of pleurisy. I injected nine ounces glucose, repeating in 48 hours. This animal made a slow recovery, but later was "put down" for tuberculosis.

This cow had four glucose injections (nine ounces) every two days. She was ailing thirteen days before she showed signs of recovery.

No. 9.—This cow was ill from April 20th to May 30th. She was dosed frequently with 9 ounces of glucose—21st, 23rd, 29th, May 1st, 3rd and 10th. There was an occasional day or two of improvement after injection. On May 10th she also received a womb injection and commenced to improve from that day.

No. 10.—This case had milk fever on April 21st. She retained the after-birth and the uterine discharge cleared up without causing trouble.

On June 6th she was suffering badly from acetonæmia. She received a uterine injection on June 6th and made good improvement. On the 16th the injection was repeated though she hardly required it.

The injection given was ac. boracic 1 ounce, iodoform 2 drams, in a quart of hot water, allowed to cool.

**Discussion.**

The President (Mr. G. Lockwood) said that he was sure they were all greatly indebted to Mr. Hudson for so kindly coming down to Lincoln to give them that most interesting paper on such an obscure and little described disease. (Hear, hear.)

In a good many of the text books it was difficult to find any reference to it at all, and on that account they should be especially grateful to Mr. Hudson for giving them such a detailed description of the symptoms and of the treatment which he had found so beneficial.

In the early part of his paper the essayist was rather inclined to have faith in the uterine treatment, and the case he described right at the end might be said to afford justification for that faith.

He (the President) was afraid he could not contribute more to the discussion, because he had not recognised many cases of acetonæmia; he might have had them, but he had not recognised them as such.

Mr. W. W. Lang expressed his indebtedness to Mr. Hudson for visiting the Division for the purpose of contributing that exceedingly valuable paper. The Division had recently tried to get out of Mr. Hudson's debt a little by making him an Honorary Member, but he had pushed them even further into that debt than they were before. (Hear, hear.)

The treatment he (Mr. Lang) had adopted throughout, as an Imrie-ite, was the use of formalin with sodium bicarbonate added, with very good results. He had found that a case of acetonæmia, if observed early, was quite easily cured by that method, but if the case had been in existence for a week or ten days he found it most difficult to cure. On one occasion he tried injecting Lugol's solution into the uterus, but it did not seem to have any effect.

He had expected to find the disease during the winter months, yet at the present moment he had two cases which had perplexed him very much. One, he was glad to say, was better, and to that he gave calcium subcutaneously. The cow went down with fits, but was responding well to treatment. In the second case the symptoms to which Mr. Hudson had referred rather made him conclude that the cow was suffering from acetonæmia; she was off her food and looked a little dazed, but he had not noticed the acetone smell. However, he did not expect to find acetonæmia at this time of the year, therefore he had not put the condition down as such.

What Mr. Hudson had told them would be a great help to them—certainly to him, at all events—and would greatly increase their knowledge of these cases.

The Hon. Secretary (Mr. H. C. Reeks) observed that neither Mr. Hudson nor Mr. Lang had mentioned that when they used to call these cases post-parturient dyspepsia ordinary udder inflation used to work very well. It was obvious, however, that those cases were not cases of milk fever.

Captain A. J. Hines observed that he had had a number of cases this year which it now appeared to him might have been cases of the nervous form of acetonæmia, although he did not realise it at the time. They puzzled him very considerably. They occurred in cows out at grass, in rather poor condition and recent calvers. When he was called to them, they were usually down and in convulsions. Considering that the condition was in some way due to calcium deficiency, he inflated the udder in each case and they responded to the treatment, apparently recovering in a few hours' time. They would go on all right for about a fortnight, then there would be a recurrence of the symptoms and death would result quite suddenly. It was true that he did not notice the smell of acetone in the breath of these animals, but that might be because they were out at grass.

In the last case he had the convulsions were most violent and it was with the utmost difficulty that he was able to inflate the udder. The case followed the same course as each of the others and died about 10 days later after having apparently recovered from the first attack.

Captain K. J. S. Dowland observed that he had had one or two cows with a condition which he was afraid he did not diagnose as acetonæmia, but which he now thought was probably at least related to it. Before proceeding to describe these cases, however, he would like to ask Mr. Hudson whether or not he had evidence that he was dealing with a pathological entity. He should have imagined that acetone in the blood could accompany many diseases, *e.g.* a septic focus in the uterus; an endometritis, as the result possibly of the retention of cleansing; or he could imagine it accompanying indigestion or occurring as part of an ordinary dietetic acidosis. In horses he considered that one got "dietetic acidosis" quite commonly; they came out all over lumps, very much like the urticaria of man. He found a great deal of it in India, and he put these cases on to sodium bicarbonate, which he found acted like a charm when given in fairly large doses. That, he thought, lent confirmation to the theory that the condition was an acidosis, and he could imagine acetone being present in the blood in some such cases. He had a half-bred Guernsey cow, which was in an irritable, nervous condition. She had always been a

highly strung animal. At the time of examination just a movement made her "jump out of her skin." He noticed no smell of acetone, but he was not looking for it. He had not got his milk fever apparatus with him, so she was not pumped up, and the next day she was dead.

A short time ago he had a case of fits in a cow which had been calved three or four days when she had her first attack. She had recovered from the fits when he got there. He inflated the udder, gave a dose of cardamon and for eight or nine weeks she went on all right. Once again he noticed no acetone, though he was not looking for it. He went down again when he was called and found her in epileptiform convulsions. He blew her up, and left some chloral hydrate to be given. She got up in six or seven hours and walked back from the field to her stable and when he got back to see her again that evening she was apparently normal. Unfortunately, he was unable to follow up the case because the owner had the cow slaughtered.

Had the essayist ever noticed, as an accompanying symptom of acetonæmia the condition sometimes spoken of as laminitis in cattle after calving? He had never been able to account for it, but it had struck him forcibly on occasion that he had seen laminitis in the bovine, which was supposed to be rather rare.

Mr. R. Swaby enquired if Mr. Hudson had found that a large purgative dose often precipitated sudden death in these cows; also, was a discharge from the uterus constant in these cases.

Captain J. W. Beaumont stated that he had had a few of these cases but they had all died; no matter what one did it seemed that one could not save them. He always gave calcium treatment intravenously, udder inflation and a strong purgative, but although they might recover for a few hours they died very suddenly at the finish—one did not know what to do to keep them alive.

Mr. R. Hudson enquired if any member had tried the glucose treatment for acetonæmia, receiving the reply from Mr. Swaby that he had used it with good effect in some animals, but with none in others.

### The Reply.

Mr. R. Hudson, replying to the points raised in the discussion, said that he thought it was rather surprising that veterinary surgeons had not recognised the condition of acetonæmia, if not as a pathological entity, at least as a disease which was striking and noticeable in dairy cattle; nevertheless he must admit that, in his own case, it passed unnoticed for a long time. Moreover, it was a disease which was certainly on the increase and was of added importance from the fact that this country was paying more attention to milk production. A veterinary friend of his, who was also a farmer and milk producer, after hearing the symptoms of the disease remarked that a cow of his had gone off in the same way, but that he did not recognise the nature of the trouble; in fact, he could not make out what was the matter. Therefore, if a veterinary surgeon could not recognise the condition amongst his own cows he was much less likely to recognise it in the ordinary way of practice. He (Mr. Hudson) certainly did think that some of these cases were very difficult to recognise, especially when they were out in the yards; he thought that when one got a cow that was not doing well, was going down in milk (and what was most noticeable was the extreme rapidity with which they wasted) it was as well to smell her breath and try her milk—during the period of calving she was almost sure to be a case.

He could not see that the Chairman was right in saying that the theory of uterine origin was rather shaky. The theory had two generations behind it; men whose views could not easily be set aside. The opinion of milk producers in his district was that it was the second cleansing which was responsible. There was no second cleansing; the cow could not cleanse completely and then go down with this disease, and his opinion was that in the cow that was closely housed that came down to calving with a heavy womb, and got no exercise to help in the removal of the fluid there, the os closed very rapidly and held back a considerable amount of fluid. That fluid might not be septic, in that it might not stink, but there might be some bacterial element which led to the formation of toxins and their absorption into the blood stream which might bring about that condition of acetonæmia. Even the glucose theory did not upset that; it was a recognised fact that the ductless glands, many of them, were readily upset by toxins produced in various parts of the body.

One case that struck him as having a great bearing on the septicæmic theory was a cow that he saw some time ago, upon which he reported before he knew anything about acetonæmia; the case was reported in the *Veterinary Journal*. This was a cow which, a fortnight before she calved, was in very heavy condition and had some stomach trouble. She became all right in a few days, however, and went on to calve, but three weeks after calving she had gone to an absolute wreck. She was stinking with acetone; her heart was beating at an enormous rate and very heavily; she was standing with her back arched and on micturition she had the peculiar movement of which he had spoken, which seemed to indicate pain in the urethra. The most peculiar symptom, however, was the condition of the eye; the lower third of the anterior chamber was full of blood and the cow was as blind as a bat—there was a hæmorrhagic dropsy of the anterior chamber, and when the cow was turned out she could scarcely see to walk. There were also multiple petechiæ and she looked like a case of septicæmia. He had seen cases ranging through all degrees from the mildest type to that very extreme septicæmic type. This particular case was treated with uterine injections—it was prior to the time at which he knew anything about glucose—and she made a good recovery. It was quite possible that the toxins had interfered with the release of blood sugar from the liver.

Mr. Lang had mentioned that he was still carrying out formalin treatment and that in some cases he had found it of benefit but not in others. He (Mr. Hudson) thought that Mr. Lang's experience in that respect coincided closely with his own early treatment of the disease, for he, too, had no success with formalin in advanced cases. He believed that cases of the nervous type had shown benefit through injections of calcium; probably those cases which exhibited nervous symptoms, as well as having a deficiency in glucose had also a deficiency in calcium. They reacted to calcium injection and also to udder inflation, where they had an increase in the calcium in the blood as the result of inflation of the udder. Apart from the cases that showed nervous symptoms, he had never seen any that derived any benefit from udder inflation. He thought it just possible that for a few hours after inflation the animal derived benefit from the increase of oxygen, but in his opinion it had not the slightest influence on even a moderately severe case where there were no nervous symptoms.

Mr. Hines had mentioned a case which was probably one of acetonæmia, in which the subject had fits and died, but in which the fits were relieved by udder inflation. He (the essayist) thought that point was dealt with in his remarks on udder injection in general. It was quite possible to get cattle showing fits independently of acetonæmia.

The question had arisen as to whether the condition with which he was dealing was a pathological entity. Acetonæmia certainly was not; he had remarked in his paper that acetonæmia was only a symptom; it was not a disease. Acetonæmia could occur along with milk fever very strongly; in fact, so strongly, he was certain, as to have an influence on the disease of milk fever. It could occur in heifers previous to calving; it could occur in tuberculous cows and in cows with dead calves in the uterus. Granted that they could have acetonæmia in so many conditions (and one other particularly interesting condition was traumatic pericarditis), yet, there was no reason why they could not regard it as a pathological entity associated with some condition within that period after calving.

It was difficult, if they did not know what actually

was its origin, to say that they had got the disease in any particular case. He had noticed it several times associated with milk fever, but it cleared up when the cow recovered; therefore he did not think they could consider it as a condition that was carrying on through milk fever, with which, in his opinion, it had nothing in common. He believed there were cases of milk fever which might carry the acetonæmic condition, and those cases readily recovered with udder inflation, but the primary condition was one of milk fever and when that was cured the acetonæmia vanished. The discussion on acidosis, urticaria and acetonæmia was one which one could not very well clear up. It had been recognised by Continental observers that there was an acidosis in connection with acetonæmia, and from the symptoms he was inclined to think that that was the case, but he had never seen anything resembling uticaria in cattle associated with acetonæmia. The blotchy condition one saw in cattle, in which they got swelling of the nostrils, eyelids, vagina, etc., was the nearest approach to urticaria that he had seen in cattle; it was a similar condition to that seen in the horse, but he would not regard that as an acidosis. If it (the acetonæmia after calving) were only a simple acidosis, he did not see why large doses of sodium bicarbonate should not easily relieve the condition, but it did not, even in doses of ʒviii per day.

A member had quoted a case showing epileptiform symptoms, which recovered with udder inflation: that was what he had admitted in his previous remarks.

He had never come across a case that had shown the least sign of laminitis. One did see cases of laminitis in cattle after calving, but he had never seen laminitis associated with this disease, nor had he heard it mentioned as a symptom.

In reply to Mr. Swaby, in his (Mr. Hudson's) cases the os was tightly closed and there was virtually no discharge—the merest trace of a white, glairy fluid like the white of an unboiled egg. There had been such an early and complete closure of the os that fluids must be retained.

He had not found that excessive purgation precipitated sudden death in these cases: he could not see that purgation would influence the disease in any way.

The President observed that he desired to correct a wrong impression that he had evidently given the essayist, *viz.*, that he thought that his (the speaker's) confidence in the uterine treatment had been shaken. He (Mr. Lockwood) must have expressed himself very badly, because he certainly thought that uterine treatment was a very important part of the treatment of acetonæmia.

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### The Royal Sanitary Institute.

A visit of the members of the Royal Sanitary Institute has been arranged to the new Abattoir, Meat and Cattle Markets at Liverpool on Saturday, November 21st, at 2.30 p.m. A description of the Abattoir and Markets will be given by the Chief Food Inspector, Mr. J. D. Allen.

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### British Horsemen in America.

A British Army team, consisting of Captain A. L. Cameron (Royal Horse Artillery), Lieutenant J. A. Talbot-Ponsonby (7th Hussars), Lieutenant F. R. W. How (8th Hussars), and Lieutenant M. P. Ansell (5th Inniskilling Dragoon Guards), is visiting America, at the invitation of the American and Canadian Governments, to take part in the horse show competitions being held this month.

The results of the jumping in the Boston Show were as follows:—International Military (Individual) Competition: (1) Lieutenant J. A. Talbot-Ponsonby. International Military Team Competition: British team held first place with five teams competing. The same team came in first and fourth in the open team competition, in the later instance on different horses.

# Clinical and Case Reports.

## A Peculiar Case of Ascites in the Chow.

A. R. Smythe, F.R.C.V.S., D.V.S.M., Falmouth.

*Subject.*—A blue chow bitch, about six years old, which had been in the owner's possession for six months; the former history of this dog was unknown.

The animal was brought for treatment, suffering from a pendulous abdomen, due to ascites.

Clinical examination failed to reveal any very evident pathological cause. Paracentesis was performed and the bitch progressed for six weeks, when the fluid began to collect again.

Treatment was continued for about twelve months, but the periods between the enforced removal of the ascitic fluid became progressively less.

Towards the end of this period, two very enlarged ovaries became increasingly apparent to digital manipulation and as emaciation was becoming pronounced, the bitch was destroyed.

On each occasion on which paracentesis was performed, the amount of fluid removed ranged from four to six pints and in colour it approximated to normal blood.

Drawn into a shallow pan, attention was from the first attracted by the presence in the ascitic fluid of peculiar bodies. In the warm fluid, these bodies were of variable shapes, mostly round or ovoid, and they were motile in that they continually changed their contour. In colour, they were translucent or pearly grey and in size they ranged up to that of a small pea.

On cooling, they became definitely round or ovoid and more opaque in colour.

Under the microscope they showed an indefinite structure, and with acid, they gave evidence of calcification

The accompanying photograph shows the two ovaries after removal and a number of the peculiar bodies present in the ascitic fluid.

After *post-mortem* the two ovaries were removed and sectioned and their histology showed them to be involved in a papilliferous cystic carcinoma, a condition also met with in the human ovary. On *post-mortem* the abdominal cavity contained a large quantity of hæmorrhagic fluid, which held an immense number of the above-mentioned bodies.

The peritoneal covering of the abdominal walls showed a number of the bodies adherent; many of these could be removed by wiping, whereas some were slightly embedded and more firmly fixed.

The abdominal diaphragm was thickly coated with superimposed layers of the bodies, the larger ones being very firmly embedded, wherefrom their size gradually diminished towards the surface, giving one the impression that the deeper ones had given rise to the more superficial by a process of budding.

The covering of the pericardium also contained a few of these bodies, whereas the remainder of the pleural cavity was free.

**Discussion.**

The pathological explanation of this condition is obscure.

On clinical evidence, the ovarian condition would appear to be a late change, possibly aggravated by the ascites.

The bodies described were submitted to several well known parasitologists, who were unable to classify them with any of the known forms of parasitism.

In the *Révue Vétérinaire,* 1906, Sendrail and Cuillé have stated that they have observed a number of cases of ascites in the dog, in the neighbourhood of Toulouse, due to the presence in the peritoneal cavity of a multitude of parasites. These parasites were cystic in form and of an undetermined nature, varying in size from that of a pin's head to that of a pea. They describe cases in which the condition has been transmitted to healthy dogs by the peritoneal injection of ascitic fluid taken from dogs affected with the disease.

They further state that Neumann, 1896, appears to have established that the Dithyridium is a larval form of a tænia, *Mesocestoides lineatus*, inhabiting the intestine of the dog, and they think the cyst they have recorded is of the same origin, *i.e.* a larval form of the same cestode, arrested and fixed in an incomplete, evolutionary stage.

## A Few Notes on Milk Fever.

**T. G. Heatley, O.B.E., M.R.C.V.S., Woodbridge.**

I have recently had four deaths following attacks of milk fever; the animals regained consciousness, but did not get up, appetite was in abeyance and there was practically no action of the bowels, death occurring in two or three days.

*Post-mortem* examination revealed the livers to be abnormal in all the cases. Two showed considerable enlargement with a pale fatty appearance; the other two were cirrhotic. The peculiar coincidence was, however, that they all showed evidence that at some time there had been injury by a foreign body from the stomach, as in each case the stomach, liver, diaphragm and lungs were firmly adherent to each other by fibrous tissue and in no case could I get a history that the animal had at any time shown any illness.

All my life I have made it a practice to make *post-mortems* on fatal cases, where the cause of death was not obvious, and certainly in 90 per cent. of fatal cases of milk fever the liver has been abnormal. Possibly the abnormal condition of the liver in the above cases may have had its origin in the injury caused by the foreign body.

I desire to relate another interesting case of milk fever where recovery was slow; the animal getting up, but the appetite remaining precarious, and the bowels very torpid for three or four days. My diagnosis to the owner was that I suspected that some foreign body had perforated the stomach, probably during parturition pains.

On the fourth evening she seemed comfortable, but during the night developed a most painful mastitis, the withdrawn material having the appearance of melted butter-fat, rather more noticeable because she was a Guernsey. Culture of the material demonstrated that the mastitis was due to *B. coli* and I gave it as my opinion that most probably the suspicion of a foreign body piercing the stomach was correct, and that the mastitis was due to invasion of the blood stream by the coli organisms.

I had not the opportunity of seeing the finish of the case, as someone came along and bid a price for her as she was. I have lost sight of her.

### Silver Fox Exhibition.

"The Silver Fox Exhibition which opened yesterday at the Agricultural Hall is convincing proof that the new industry of silver fox breeding has become firmly established in Great Britain," says *The Times* of November 5th. "There are now 115 breeders in the country with about 60 farms situated in all parts, from the extreme north of Scotland to the southern counties of England. At this year's exhibition, the sixth annual show, but the first to be held in London since 1926, there are 201 entries from 38 breeders.

"The silver fox is a black animal, and for purposes of exhibition there are seven classes—black (without any silver), slight silver, quarter-silver, half-silver, three-quarter silver, pale silver and extra pale silver." . . . .

"Judging is, of course, a task for experts, but at the same time it affords much interest to the less enlightened looker-on. The competing foxes are brought to a long, narrow table, across which they are held by two men (or women) standing each on one side of the table and holding the front legs and back legs respectively of the fox, which has previously been muzzled with a piece of plain black tape. In gauging the quality of the skin and fur the judge blows gently into the pelt and gives an expert touch to brush or tail.

"A prize pair of foxes at the show is said to be worth from £150 to £200."

"From a small body of eight who started the Silver Fox Association six years ago, that Association has grown to a membership of 116, and has now issued its first Stud Book," says *The Farmer and Stockbreeder.* "It is expected that this will give complete confidence to the prospective buyer and discourage the idea that it is necessary to go outside this country for the good class of foundation stock which is absolutey necessary if a proper beginning is to be made. In the six years during which the Association has been operating it has registered over 2,000 British-born animals. An important rule is that inspection is necessary in addition to a full pedigree record before an animal can be admitted to the Stud Book, and it is learnt that the standard set by the inspectors is high."

# Abstracts.

**[Un nouveau médicament pour le traitement des piroplasmoses. (A new drug for the Treatment of Piroplasmoses.)** THEILER, A. *Bull. Soc. Path. Exot*, 1930. May 14, Vol. 23, No. 5, pp. 506-529. With 22 text-figs. Ex. *Ind. Jour. Vet. Sci. and Husb.*, Vol. 1, Part II, June, 1931.]

The author points out that trypanblue has had a wide application in the successful treatment of piroplasmosis both natural and experimental. But, more particularly since the war, there have appeared on the market substances called trypanblue which not only are devoid of any parasiticidal action, but are liable on injection even by experienced persons to produce alarming symptoms of shock. In the course of a tour in Queensland two years ago Theiler heard it said that trypanblue was quite useless for the treatment of red-water as it occurred in that country.

This fact caused the author to decide upon the publication of the results which he obtained at Onderstepoort in 1925 and 1926 with a new drug which he calls "Piroblue." The experiments carried out in the Sandoz laboratories by Muller have shown that the parasiticidal action of stains such as trypanblue and others can be greatly increased if they are combined in an appropriate form with the bile acids. This combination can be effected without producing any proportional increase in their toxicity.

The bile acids and their salts possess no parasiticidal power of their own, but their association with pure trypanblue renders it possible to reduce the dose of the drug to a point at which toxicity does not come into consideration at all. Not only so, the parasiticidal action is actually increased.

According to Stoll, the explanation of this effect is probably as follows: Bile acids exert a profound effect upon the surface tension of liquids containing them. This fact is made use of commercially in dyeing, and this property enables drugs combined with them to come into closer contact with parasites.

Two compounds, which are called T18 and T19 were used for experiment. These were composed of the pure dye and sodium cholate in different proportions. These compounds possessed similar properties, and it was decided to make one intermediate in composition between these for commercial application.

The toxicity of "Piroblue" was tested upon mice, but, in spite of the use of a large series of animals (nearly 100) the results were, for some unknown reason, discordant.

A series of guinea-pigs was placed under test by intravenous injection. In the majority of cases a five per cent. solution was used, and doses were graduated so as to give 0·1 g. per kg. upwards. In this series concordant results were obtained, and the toxic dose by intravenous injection was found to be 0·19 g. per kg.

Full details are given of twelve observations carried out on pure-bred Polled Angus cattle, five upon African native bred animals obtained from areas free from piroplasmosis, and five upon horses. Four of the latter were animals undergoing hyperimmunisation against horse sickness, the other a sporadic case. One of the animals was infected with *P. caballi*, and the other four with *Nuttallia equi*. The whole of the seventeen bovines used were infected artificially with *P. bigeminum*. Of the twelve Scotch animals six developed hæmoglobinuria as did also two of the African cattle. The blood used was therefore of considerable virulence. The majority of animals also developed high fever.

Both compounds T18 and T19 were used, in 1 and 2 per cent. solutions, and doses of 100 c.c. for adults and 50 c.c. for calves were given intravenously. In no case was any evidence of shock observed. One of the African cattle was given 50 c.c. of 4 per cent. solution of T18 without producing trouble of any kind.

In *every case* the temperature promptly fell and parasites disappeared from the circulation within 24 hours, and there was a *rapid* and *surprising* improvement in condition. In one case death occurred, but the calf in question should not have been put under treatment, as it was suffering from a paratyphoid infection.

T18 and T19 do not produce complete sterilisation as is shown by the fact that in eight instances parasites re-appeared in the blood in small numbers some days after their first disappearance. This is not an undesirable result, because premunisation against piroplasmosis depends upon the persistence of the parasite in the blood. The new drug is suitable therefore for use in the premunisation of animals, and it can be given without risk to valuable imported animals. The possibility of using solutions up to 4 per cent. strength permits of a reduction in the amount of liquid injected to reasonable proportions.

Until further experimentation has furnished fuller information, Theiler recommends the following doses:—

For calves or slight cases:—

50 c.c. of a 2 per cent. solution in water.
or 100 c.c. of a 1 per cent. solution in water.

For serious cases in adult animals:—

50 c.c. of a 4 per cent. solution in water.
or 100 c.c. of a 2 per cent. solution in water.

All the animals treated in the experiments had been infected by the injection of virulent blood, but the author sees no reason why similar results should not be obtained in naturally contracted cases.

The four cases of equines infected with *Nuttallia equi* were all cured, but Theiler recommends caution for the time being in drawing conclusions as to the efficacy of the drug against this parasite. The first case treated was a severe one and two injections were necessary to cause the disappearance of the parasites. In the other cases the disappearance of the parasites was not immediate, and the temperature remained high for 24 hours after the injection. It must, therefore, be concluded that the drug is probably less active against Nuttallia than against Piroplasma. The fact that a case of nuttalliasis with hæmoglobinuria recovered after two injections is sufficient to recommend the use of the drug, more particularly because up to the present no known drug has been of any value for the treatment of this condition.

It is suggested that Piroblue will probably prove effective against canine piroplasmosis and against diseases caused by Babesiella, but the author notes, without giving any

details, that it is quite without action on *Theileria parva* and Anaplasma. (Reprinted from *Tropical Veterinary Bulletin*, Vol. 18, No. 4, December, 1930.)

[**Difficulties of Diagnosing Plague in Rats.** Levinthal, W. *Zeit f. Hyg. u. Infektionsk.*, May 22nd, 1931. p. 433. Ex. *Brit. Med. Jour.*]

The author points out that, in the diagnosis of plague in rats, not only may the pseudo-tubercle bacillus give trouble, but also *B. coli*. He has recently examined a strain, identified as a plague bacillus, recovered from an epidemic among rats on a ship, in which apparently 68 out of a total of 182 rats died. In broth cultures this organism showed well-marked bipolar staining; it proved fatal to guinea-pigs in four days, though it was non-pathogenic grey rats. It was agglutinated almost to titre by an anti-plague serum. Closer examination, however, revealed that it was motile, that it produced gas in sugar media, and that it formed indol. The agglutinability of this *B. coli* strain by an anti-plague serum was found to be due to the fact that the serum had been prepared by injection of a horse, and that the agglutinins for the coliform bacillus were not specific agglutinins for *Past. pestis*, but natural agglutinins for *B. coli*. The strain was agglutinated by an antipneumococcal serum prepared from a horse, but not by one prepared from a rabbit, thus confirming the truth of this explanation. The author states that he has met with several recently isolated strains of *B. coli* which produced a plague-like disease in guinea-pigs—buboes embedded in hæmorrhagic œdema—and which in the organs closely resembled the plague bacillus. Such organisms are not infective by the cutaneous route, and rapidly lose their virulence.

[**Tests of the Efficacy of B C G Vaccine in Swine.** Jundell, I., and Magnusson, H., *Skan.: Vet.-Tidskft*; 1931, 9, 271-288.]

[The interest of the following abstract is enhanced by the publication, in the *Veterinary Record* of October 31st, of the paper by Mr. J. J. Dundon, M.R.C.V.S., entitled "Tuberculosis in Swine."—Ed., *V.R.*]

There is reference to little work on the immunisation of swine in the literature relating to B C G vaccine. On the other hand, tuberculosis in swine is important since these animals are subject to infection by the three types, of which the bovine is the commonest and the most dangerous. The chief route of infection is by the digestive tract. Reference is made to the work of Jerlov (1926) who vaccinated one pig which later, together with one control, were fed with tuberculous bovine lung tissue on six occasions. When slaughtered both showed many lesions but no conclusions were drawn. The work of Ascoli and Sanz is also annotated.

The authors carried out the following experiments. Twenty-four young pigs, certified tubercle-free, were divided into three groups of eight in each group. One group, when 14-16 days old, were injected hypodermically at the base of the neck with 10 mgr. B C G culture suspended in saline.

A second group received a total of 30 mgr. B C G culture by stomach tube. When 4-6 days old they received 10 mgr. which was twice repeated at three-day intervals.

A third group acted as controls.

The housing and feeding was arranged with special precautions against extraneous infection. All the pigs, with the exception of two of the controls, were infected *per os*, seven to eight weeks later, with a single dose of an emulsion of tuberculous bovine udder tissue estimated to contain 2,000 million bacilli per dose (method of Skar) The *post-mortem* findings are tabulated and provide invaluable data regarding the degree and site of disease processes following infection on a given date by a calculated dose of an organism of known type.

The conclusions are summarised as follows: "All the vaccinated animals developed tuberculosis within 19-21 weeks after being infected. The changes were almost as progressive as those in the control animals. The most that can be said is that the vaccination delayed the infection to a small extent." Then follow some records of weights of the pigs.

It might be thought that the dose of virulent material was too high, but compared with figures relating to this laid down by the Royal Commission, this is not so. The potency of the vaccine used cannot be called in question, since part of the same batch was used about the same time on calves, a characteristic vaccination nodule developing at the injection sites.

G. B. B.

# Review.

[**The Meat Industry.** By Anthony, D. J., M.R.C.V.S., D.V.S.M. (Vict.), and Blois, W. G., M.B.E. Baillière, Tindall and Cox. Price 7s. 6d.]

The necessity for a second edition of this small book shows that students of the Butcher's Institutes have found the contents useful information. The book is made up of two parts.

Part I.—This consists of eight chapters, deals with The Shop; Weighing; Meat as a food; Breeds of cattle, sheep and pigs; Production and distribution of meat; Preservation of meat; By-products and small goods; and Laws applicable to the meat trade.

The information supplied and advice given are certainly very useful for the young student desiring to learn the trade of a butcher. Another book with somewhat similar contents lately appeared and it must be difficult for students to decide between two books giving much the same information.

The President of the National Federation of Meat Traders Association, who should be in a good position to judge, in a "Foreword" states "no one textbook can, within its pages, contain a written account of the accumulated experience of all the working in various branches. The book, however, does make a valuable contribution to the literature on the subject."

Part II.—This part consists of four chapters dealing with The structure of beasts; Slaughter and dressing; The carcass and Diseases of animals. The structure is, considering the brief description given, lucidly described. On the subject of slaughtering considerable attention is given to the much-discussed methods of slaughter in relation to "splashing," *i.e.*, petechiæ or small hæmorrhages in

the muscular tissues. There will, no doubt, be readers who do not agree with all the opinions expressed and there are errors due probably to the fact that the author has not personally observed the method of slaughter, *e.g.*, in describing the "box method" it is stated that the pole-axe is used. In the large exporting countries generally, a hammer with a flat head is in use—not the pole-axe. Similarly in the same countries sheep are killed by the Jewish method and not stuck. In the description of slaughtering by electricity the method is reported on as unsuitable. Attention is drawn to an improved method mentioned in the *Veterinary Record* of last week's issue. The meat inspector in the district where the method is in operation has stated that he has never seen any "splashing" in the pig carcasses.

The various diseases of animals are very briefly described. It is doubtful if such description may do more than merely stimulate a desire on the part of students to know more about the subject of disease. Even if it does that it will achieve a very useful aim, for all butchers should be able to recognise abnormalities in a carcass or its organs and, in accordance with the Public Health Regulations, call the attention of the meat inspector to the condition.

The animal diagrams in the book are mere outlines with lines showing the "joints" into which the carcasses are divided. Actual photographs of sides of beef, mutton, pork and veal with the lines in colour would have been an improvement.

The printers have done their part of the work excellently. The paper is good and the print is clear. The book can be confidently recommended to the student for whom it is intended and students in veterinary science might read Part II with benefit to themselves.

T. D. Y.

### The Royal Show.

The Council of the Royal Agricultural Society of England, at a meeting on November 4th, nominated Lord Mildmay of Flete to succeed Sir Arthur Hazlerigg in the presidency of the society in 1932. Sir Arthur mentioned that Lord Mildmay's presidency would coincide with the holding of the show at Southampton. To rumours that, owing to the difficult times through which the country is passing, the society contemplated not going forward with next year's show he gave an unqualified contradiction and said that the date agreed upon was July 5th-9th, and the site of the show would be Stoneham Park.

The Council decided to support the Hunters' Improvement Society in asking the War Office that, instead of cancelling on the ground of national economy the whole of the £30,000 usually allocated for premiums for light horse breeding, the amount should be reduced to £8,000. Sir Walter Gilbey, president of the Hunters' Society, said that if the War Office could see its way to make an allowance of £8,000 they would be able to carry out the 1932 show. The abandonment of the grant entirely would press with great hardship upon all breeders of half-bred light horses and stallion owners.

The first stage of the (Licensing of Bulls) Act has come into operation. For the quarter ended September 30th, 61 rejected bulls were landed, of which 58 were slaughtered at the landing places, the other three having come in before the regulations came into force.

# Administrative Report.

## County of Renfrew.

**Annual Report of the Veterinary Officer for 1930.**

This report illustrates the extensive and varied duties which an increasing number of Local Authorities are asking their Veterinary Officers to undertake. The Local Government (Scotland) Act, 1929, has extended to several small Burghs the authority of the County of Renfrew in Public Health Acts, and their Veterinary Officer now undertakes much more meat inspection than the majority of his County colleagues. A striking benefit from veterinary supervision of slaughterhouses is noted very clearly when Mr. Abbott states that two calves killed in the slaughterhouse of an independent Burgh in the County, were reported to be affected with congenital tuberculosis. The dams were traced and slaughtered under the Tuberculosis Order, 1925, the *post-mortems* revealing tuberculosis of the uterine mucous membrane. Veterinary Officers controlling all slaughterhouses within their area would be able to follow up any cases of tuberculosis very expeditiously and reports of this disease in young bovines and pigs from the large Burghs would assist undoubtedly in finding cases of tuberculous mastitis.

A percentage of annual reports are made tedious by reason of the mass of uninteresting statistics and "copy" written round them. This fault cannot be attributed to the one under review, as the report itself and the appended tables contain only what is of real interest to anyone interested in the work. Mr. Abbott has noted specially certain items of the past year and the only fault one can find is that he has erred on the side of brevity in not "writing them up," as his opinion and deductions would undoubtedly be of value.

Some of these points are:—The value of the inspection of exempted herds with only a few cows; producers find it more difficult to keep within the coliform, than the Bacterial Count, standards of cleanliness; benefits of water-bowls; the indiscriminate feeding of young stock on the milk of diseased, or suspected, cows; sad lack of suitable facilities for isolating ailing animals; provision of powers to compel Local Authorities to furnish information of cases of tuberculosis found in their slaughterhouses; advantages of veterinary surgeons giving lectures to stock owners on preventative veterinary medicine (as practised by Mr. Abbott); the difficulties of marketing tuberculin tested milk; *post-mortems* made on reactors from tuberculin tested herds.

Other points showing the high standard of Mr. Abbott's work are:—The large number of Renfrew producers figuring in the prize list of a clean milk competition conducted by a Glasgow Co-operative Society; of 89 animals slaughtered under the Tuberculosis Order, 1925, 47 per cent. were "not advanced," 57 per cent. were reported by the County Veterinary Officer and in 26 cases the diagnosis was completed by finding *B. tuberculosis* in sputa.

It would, I think, be an improvement if Mr. Abbott gave in some of his tables the figures for previous years, as comparison would show how his well-organised work is improving veterinary public health in Renfrewshire.

F. A. D.

# N.V.M.A. Divisional Reports.

## Western Counties V.M.A.*

### Double-intradermal Tuberculin Test Demonstration at Taunton.

The Autumn Meeting of the above Division was held at the Great Western Hotel, Taunton, on Thursday, October 15th, when the following members signed the attendance book :—Messrs. Townson (President), Thorburn, Lansley, Kauntze, Parsons, Blackwell, H. Keeling Roberts, Percy, Irvine, E. R. Edwards, Bloye, R. Barons Nelder, A. G. Saunders, E. B. D. Hall, L. W. Bovett, Roach, G. A. Roberts, J. M. L. Penhale, Matthews, E. C. Bovett, Masson, R. H. Smythe, S. J. Motton, and Majors Ascott, Wright and James. Visitors :—Lieut.-Colonel P. J. Simpson (President, N.V.M.A.), and Messrs. R. E. Glover (Cambridge), J. C. Adams, H. Macdonald, H. C. Wilkins, W. H. Chase, C. V. Watkins, J. C. Hally and Major Rankin.

Apologies for absence were received from Professor Hobday, Major Fyrth and Messrs. Stephens, Harvey, Penhale, Smythe, W. B. Nelder, C. Crowhurst and J. G. Crowhurst.

Before dealing with the ordinary business of the Meeting the President (Mr. W. H. Townson) said he wished to extend a very hearty welcome to the visitors, making special reference to the delight experienced by all at the visit of the President of the N.V.M.A. (Lieut.-Colonel P. J. Simpson), and at the presence of Mr. R. E. Glover, who was to give a demonstration of the double-intradermal tuberculin test.

Lieut.-Colonel Simpson, in acknowledgment, observed: I would like to say that it is a great pleasure to come to this meeting of the Western Counties Division. During my year of office I hope to get around as many of the branches as possible, and I hope that at the other Divisions there may be as good a number of members present. I congratulate the President on the attendance.

I hope as many as possible will turn up at the Congress at Folkestone, at which I trust the attendance will be a record one. I may say an additional incentive to my visit was the knowledge that Mr. Glover was to give his demonstration, so that I might see his readings of the intradermal tuberculin test.

*Election of new member.*—Mr. C. V. Watkins, D.V.S.M., M.R.C.V.S., of the Seale-Hayne College, Newton Abbot, was unanimously elected, on the proposal of the President, seconded by Mr. Masson.

Correspondence.

A circular letter which had been received by some of the members from an insurance company *re* contracts for veterinary services, was considered, and for the present it was decided that no action be taken.

Correspondence from the N.V.M.A., dealing with the free treatment of animals of the poor, was laid before the meeting. A committee consisting of the Council of the Division (with power to co-opt other members) was set up to go into the matter, and report to the members of the Division as soon as possible.

*Received for publication October 23rd.

A resolution from the Midland Counties Division, forwarded by the N.V.M.A., with regard to the compulsory notification of Johne's disease, was dealt with, and a proposal by Major Ascott, seconded by Mr. E. C. Bovett, was carried, to the effect that, although it would be desirable to make the disease notifiable, the present was not an opportune time, and that further consideration of the matter should be deferred for twelve months.

### The Double-intradermal Tuberculin Test.

Prior to the demonstration of this test by Mr. R. E. Glover, B.Sc., M.R.C.V.S., from the Institute of Animal Pathology, Cambridge, the demonstrator made a few remarks on the technique and interpretation of the test. He emphasised the advisability of adhering to the recommended dose, *viz.* 0·1 c.c. of concentrated tuberculin, for both the sensitising and diagnostic inoculations, and also warned his hearers against the practice of omitting the second injection in those cases where there was no reaction to the first dose. He also stressed the desirability of using needles of adequate length and illustrated the differences in the amount of reaction with long and short needles respectively, pointing out that the most satisfactory results were obtained when the tuberculin was introduced into the deep layers of the derma.

The method of examining and classifying the swellings was then discussed, with special reference to the interpretation of so-called "doubtful reactions." In conclusion, Mr. Glover said that he wished to express his very sincere thanks to Mr. Saunders (Taunton) for his willing co-operation. Mr. Saunders had not only secured a herd for the demonstration, but had undertaken the whole of the preliminary injections and he was extremely grateful to him for his able assistance.

The members now proceeded to the farm, where twenty animals had been tested with tuberculin and glycerine broth. The results were explained by Mr. Glover.

Discussion.

Mr. R. H. Smythe thought the demonstration proved that the interpretation of results was not always so very simple. The fact that there might be a difference in results by the use of different brands of tuberculin could lead to difficulties; one might get a normal reading with a thin tuberculin, and a normal, but different, reading with a thick tuberculin. Manufacturers had informed him that the cause of variations in the thickness of tuberculin was a difference in the glycerine content of different brands.

There should be a standardisation of the glycerine content and the medium, as well as of potency.

Some co-ordination of opinion as to what was a reaction, was necessary: a cow might be passed by one man, and condemned by another on the same measurements a few weeks later. That should not be possible. Recently in a sale catalogue he had seen measurements of 9, 14, 20 and 7, 12, 14 recorded as having passed the test.

He understood Mr. Glover to say that a reaction was to be gauged by the character of the swelling only: he did not agree. He thought the use of glycerine broth as a control should be compulsory, for he failed to see how the Ministry of Health could form an opinion on a chart without a control by glycerine broth. Moreover, he thought that in some cases all the phenomena of a tuberculin reaction could result from glycerine broth injections. With

regard to the sub-palpebral test, his experience was that this test picked out so many tuberculous animals that, in consequence, many dealers refused to have it applied. He (Mr. Smythe) erred on the side of condemning doubtful reactors. The eye test picked out some which he would have missed with the double-intradermal test.

A control test, to be used with the double-intradermal test, should be compulsory.

Major Ascott said he appreciated the opportunity of witnessing Mr. Glover's demonstration. He agreed with Mr. Smythe as to the reliability of the sub-palpebral test. He found it of great service in cases which would otherwise have been doubtful. In two recent cases he had seen a slight reaction to both the intradermal (double), and the sub-palpebral tests. At the *post-mortem* examination he could find no evidence of tuberculosis, although he thought with laboratory facilities evidence of tuberculosis would quite likely have been discovered. What he did find in both cases was evidence of Johne's disease, and he wondered if it were possible that Johne's disease could cause a reaction to tuberculin.

Mr. Irvine asked if the caudal fold test was an accurate test.

### The Reply.

Mr. Glover prefaced his reply by remarking that he appreciated the honour of being invited to address the Division, and thanked the members for the excellent attendance.

The question of the standardisation of the preparation and control of the manufacture of tuberculin had been raised on many occasions and it was unfortunate that it had not been possible to introduce measures which would ensure that only tuberculins of known potency were placed on the market. Certain experiments, which they had undertaken, had indicated that there might be a marked variation in the strength of different brands of commercial tuberculin, and it was not improbable that some of the unsatisfactory results experienced by some practitioners were attributable to the use of samples of low potency. He urged them to obtain their tuberculin from manufacturers who carefully standardised their products. The variations in viscosity of different brands of tuberculin were probably due to differences in the amounts of glycerine, and since the swelling produced by the intradermal injection of tuberculin was largely due to the glycerine content of the product, he agreed that a standard method of preparation was highly desirable.

Turning to the interpretation of the reactions, he pointed out that it was impossible to classify them solely on the size of the swellings; nevertheless, measurements were of value, as abnormally high readings, recorded as negative reactions, frequently indicated a misinterpretation of the test. Measurements of 9, 14, 20, might conceivably occur in a non-tuberculous animal, but he thought it was most unlikely. He wished, once again, to emphasise the importance of carefully palpating the swelling in order to detect slight degrees of diffuseness which constituted positive reactions. He scarcely thought it desirable or feasible to insist upon the inoculation of glycerine broth as a control measure at every test.

He was interested in the observations of Mr. Smythe on the sub-palpebral method. Mr. Smythe had had extensive experience of this test and his observations warranted a careful consideration. He (the speaker) could only bring forward the objections to this test which had been furnished by certain Continental workers (Januske, Seiter and Blumenberg) who had shown that a proportion of tuberculous animals varying from five per cent. to 12 per cent. failed to give a local reaction to this method. He had also, in his preliminary remarks, stated his objections to the sub-caudal fold. Nevertheless, he had no desire to discourage those practitioners who preferred these tests, and could appreciate their desire to adhere to the methods which they found most satisfactory. At the same time, he held the opinion that the double intradermal test, when correctly interpreted, was an extremely exact test, exceeding in accuracy both the sub-palpebral and sub-caudal methods.

So far as he was aware, animals affected with Johne's disease would not give a local reaction to tuberculin.

*Any other business.*—Major Ascott said that, in view of the fact that members might be called on to give evidence or express opinions *re* variations in the composition of milk, they might like to obtain a very useful booklet published by the Ministry of Agriculture and Fisheries. The booklet, which formerly bore the number 65, was now Bulletin No. 26: "Variations in the Composition of Milk." Price 4d.

*Votes of Thanks.*—At the end of the meeting, on the proposal of the President, a very hearty vote of thanks was accorded to Lieut.-Colonel Simpson for visiting the Division, to Mr. Glover for his demonstration, to Mr. Saunders for the work he had done in connection with the tuberculin tests, and to the Committee of the Cotford Mental Hospital for putting the herd of cattle at the disposal of the Division.

S. J. Motton, *Hon. Secretary.*

## Lincolnshire and District V.M.A.

The autumn meeting of the Lincolnshire and District Division, N.V.M.A., was held at the Albion Hotel, Lincoln, on the afternoon of Thursday, October 8th, 1931.

The President (Mr. G. Lockwood) occupied the chair and there were also present Captain J. W. Beaumont, Captain K. J. S. Dowland, Mr. G. Eaton, Captain A. J. Hines, Captain W. W. Lang, Captain A. Logan, Mr. R. Swaby, Mr. H. C. Reeks (Hon. Secretary) and Mr. R. Hudson (Hon. Member).

The minutes of the previous meeting of the Division, which had received publication in the *Veterinary Record*, were taken as read and were signed as correct.

The Hon. Secretary announced the receipt of apologies for absence from Captain T. B. Bindloss, Mr. P. E. Emson, Mr. F. W. L. Gooch, Mr. W. Grasby, Principal F. T. G. Hobday, Mr. J. T. Holmes, Professor James Macqueen and Mr. R. T. Sheppard.

*Resignation.*—The Hon Secretary informed the meeting that, in his letter, Mr. Holmes had submitted his resignation from membership of the society, as he was giving up practice.

The resignation of Mr. Holmes was accepted with regret.

*Nomination.*—In his letter of resignation, Mr. Holmes had nominated as a member his successor—Mr. E. P. Emson—whose nomination was welcomed by the meeting.

*Treatment for Sick Animals of Poor Persons.*—The next

business before the meeting was the consideration of a memorandum from the N.V.M.A., referring to schemes for providing free treatment for the sick animals of the poor. In the absence of the relative documents, the main points of the Centralised and Decentralised Schemes were outlined by the Editorial Representative, N.V.M.A., and, after deliberation in committee, the following resolution was unanimously passed: "That the Lincolnshire and District Division, N.V.M.A., favours the adoption of the Decentralised system of treatment for the sick animals of poor persons, and stresses the necessity for the co-operation of all practitioners in any area in which such a system is established."

*Paper.*—Mr. R. Hudson, F.R.C.V.S., of Retford, was introduced by the President, who called upon him to read his paper "Acetonæmia and the Consideration of Glucose Treatment." The paper was heard with much interest, and evoked a well-sustained discussion, to which Mr. Hudson gave an exhaustive reply. [Mr. Hudson's paper and a report of the ensuing discussion and the essayist's reply, are reproduced earlier in this week's *Record*.—Ed.]

*Votes of Thanks.*—At the conclusion of Mr. Hudson's reply, the President observed that he was sure that all the members had thoroughly enjoyed that most interesting paper, and accordingly he asked them to accord the essayist a very hearty vote of thanks.

The vote was passed with enthusiasm, and a similar tribute was paid the President for his conduct of the meeting.

Before the company dispersed they spent a very pleasant social hour over the tea-tables as the guests of the President.

H. C. Reeks, *Hon. Secretary.*

## Central Veterinary Society.

### Dinner-Dance Success at the Hotel Russell.

Victory Medal Presented to Mr. Walter Perryman.

It was perhaps not an unrelated circumstance that a renewal of the policy of enlivening the Central Veterinary Society Annual Dinner with a dance (to the exclusion of speeches)—so successfully initiated in 1929, but abandoned last year—should have coincided with the first post-election appearance in the Chair of Major Hamilton Kirk. In any event, it was very evident that the President's obvious appreciation of so vivacious a mode of entry into the sessional activities of the Society was shared by the great majority of the Fellows present and, without exception, by their ladies: in fact, the very large company who participated in this delightful function at the Hotel Russell entered into the spirit of that most informal date in the calendar—November 5th.

The selection of the Hotel Russell as the scene of the fifty-eighth Annual Dinner proved an admirable choice, the management giving every satisfaction by serving a capital repast and providing a fine and spacious dancing floor.

The Loyal Toasts having been honoured, Mr. G. P. Male, M.R.C.V.S., President of the Royal College of Veterinary Surgeons, submitted the toast of "The Central Veterinary Society," saying, at the outset, that he believed he had been asked to propose that toast because he was the only one who could be trusted to make a short speech. (Laughter.) Certainly, after the plethora of speeches they had had during the last three weeks, one could quite understand the committee feeling very diffident about allowing speeches at that function, but he thought they would agree with him that on such an occasion as that, which was an annual function, it was only seemly that they should propose the health of that great Society of theirs and should wish the President success in his year of office. (Hear, hear.)

It needed no eulogies of his to commend that toast to them: their Society had great traditions of learning and philanthropy, and it numbered amongst its members so many distinguished members of their small profession that he was sure they could congratulate themselves with great reason on the great strides that the Society had made and the fact that it was still progressing. In that connection, he felt that they owed their Honorary Secretary a very great deal for all the work he had put in and the success he had made of the Society. (Cheers.)

Perhaps he might be allowed to make just one personal reference, and that was that he desired to be allowed to take that opportunity of thanking them most sincerely for the honour they had conferred upon him in electing him one of their Vice-Presidents: he had not had an opportunity of thanking them before and he would like to do so then. He desired also to thank the President and Fellows for inviting his wife and himself there that evening and entertaining them with such lavish hospitality.

When he was asked to propose that toast he accepted with the greatest pleasure, because it gave him an opportunity of saying how pleased he was and to congratulate their President on being elected to the Chair of that Society. It was an honour which Major Hamilton Kirk richly deserved, for the wealth of his energy and enthusiasm for the veterinary profession and for the Central Veterinary Society. (Hear, hear.) He was quite sure that their new President would fulfil his duties with very great success and with credit to their Society. (Cheers.)

The toast having been drunk with enthusiasm, the President (Major Hamilton Kirk, M.R.C.V.S.) in reply, said:—

Mr. Male, Ladies and Gentlemen, it is my proud privilege and pleasure to respond to the toast so kindly worded and proposed by Mr. Male, and on behalf of the Central Veterinary Society I desire to tender him our most grateful thanks. (Cheers.)

Doubtless most of you were much relieved when you heard Mr. Male tell you that the length of speeches this evening was to be reduced to a minimum. Indeed, the Council of this Society went further than that when they decided, wisely as I think, that most of the usual speeches should be deleted altogether. However, there was no escape for me—(laughter)—but I do not propose to detain you for one second longer than can be avoided. Yet I do think that I should be lacking in courtesy were I to fail to take this opportunity of expressing my sincere personal welcome and gratitude to the whole assembly for so well supporting me at the opening of my year of office.

On behalf of the Central Veterinary Society, I wish to extend the heartiest welcome and good wishes to the guests, of whom I am pleased to note we have a goodly

number. Among these guests, it may interest you to know that we have with us this evening not only Mr. G. P. Male, the President of the Royal College of Veterinary Surgeons, who has already addressed us, but also Lieut.-Colonel P. J. Simpson, D.S.O., President of the National Veterinary Medical Association; Dr. Abel Brion, Laureate of the French Academy of Medicine and a teacher at the Alfort Veterinary School; Dr. Ernest Young, late President of the Hunterian Society; Mr. Garland Wells, managing director of the Clapton Greyhound Stadium, Chairman of the London Tracks Committee and a founder and leading light of the Greyhound industry, and last, but not least, I have, sitting on my left, my father—(applause)—a well known and much respected member of the veterinary profession for nearly 50 years and a member of the English Bar for over 20 years. I am sure we are honoured that they should grace our table, and long may they come among us to add dignity and pleasure to our proceedings. (Cheers.)

It would ill become me were I to omit some allusion to the ladies and, as they are not to have a toast of their own, I would like to say how very much we mere men appreciate and rely upon their presence on occasions such as this. (Hear, hear.) Indeed, without the ladies there could be no dance; and the fact that so many wives are in evidence, obviates the necessity for their dutiful husbands rushing home at an early hour.

I feel that I must express my keen sense of appreciation of the honour the Society has done me in placing me at its head *pro tem.*; and I realise to-night, as I have seldom realised before, that to be the President of so large and important a Society of professional men, is a triumph of which one may be justifiably proud. (Hear, hear.) Some years ago it was common to hear veterinary surgeons' children ask this question:—"What did you do in the Great War, daddy?" But now that war is forgotten, it is more likely that one will hear them ask "What have you done for the Central Veterinary Society?" (Laughter.)

**Presentation of the Victory Medal.**

However, I am not the only person in this room this evening upon whom this Society has thought fit to bestow honours, and it will soon become my pleasurable duty to introduce to you the recipient of the Victory Medal.

Soon after the War this Society conceived the laudable idea of commemorating the Allies' victory by presenting a gold medal to the Fellow of the Society who, from sheer merit, had earned the approbation of his fellow members. Some very excellent men have received this medal in the past, but there can have been none more loyal, more persevering, or more deserving than the gentleman who receives it to-night. I refer to Mr. Walter Perryman. (Loud cheers.) I was sure that name would receive universal approval. There can be no more certain method of sustaining the life of a society, or a club, or any other body, than by the regular attendance of its members, and I think I can truthfully state that Mr. Perryman has earned the best record in this respect throughout his 30 years of membership. Mr. Perryman has read many valuable papers before this Society and has exhibited very numerous morbid specimens for clinical discussion, and has occupied almost every office from President downwards. I have no doubt that he has thoroughly earned this mark of our appreciation. Will Mr. Perryman kindly step forward to receive his just reward? (Cheers.)

Presenting the medal, the President said: Mr. Perryman, it is a great personal pleasure to me that it has fallen to my lot to present you with this handsome Victory Medal, and in receiving it you will have the satisfaction of knowing that there is no higher honour we can bestow upon you. I only hope that your membership of the Central will continue for another 30 years, and if it could, I am certain that your interest and activities would be just as well sustained. (Cheers.)

The recipient of the medal was accorded rousing musical honours, and the President concluded by saying:—

Ladies and Gentlemen, there remains only one more thing for me to do and that is to hope that you will have a most enjoyable evening, and that you will remember this function occurs annually, and will try to make a habit of attending. (Loud cheers.)

Mr. Walter Perryman, M.R.C.V.S., in expressing his appreciation of the bestowal upon him by the Society of the Victory Medal, said that that was a very embarrassing moment for him, particularly as their President had been so eulogistic. Of course they knew Major Kirk's abilities in the matter of eloquence, and he hoped that they would make some allowance for the occasion and discard some of the praise the President had showered upon him. (No, No.)

As the recipient of the medal, he could assure them that he appreciated it to the full, but he must disagree with the President's observations that it was the highest honour in the gift of the Society—that was the Presidentship, to which Major Kirk would do full justice during his year of office—but it was the second highest honour and he appreciated it beyond power of words to express.

He knew that a great many of the previous recipients of the Victory Medal held a high position, not only in their Society, but in their profession, and when he learned that he was to have it he really wondered why, because there were so many Fellows of the Society who were quite worthy of the honour they had so kindly conferred upon him that evening.

Their President had told them of the work the Society had done and of the interest that he (Mr. Perryman) had always taken in it, and he could honestly say that he had never ceased to regard it not only as a pleasure but as a duty consistently to associate himself with that work—(hear, hear)—moreover, he felt very strongly that if all the members of the profession would join the various veterinary societies great benefit would accrue, not only to the profession but to themselves, both socially and professionally: membership of a society such as that was a great help. (Cheers.) To his mind, there was a far better feeling amongst the members of the profession than there was many years ago, and for that happy state of affairs there could be no question but that veterinary societies were in a large measure responsible. (Hear, hear.) Professionally, it was everyone's duty to join, for by that means the older members were afforded a splendid opportunity of keeping abreast of advancements in the knowledge of medicine and of keeping in touch with all up-to-date improvements.

Sometimes he had heard it whispered that membership of a veterinary society was a good excuse for having an afternoon off. (Laughter.) He thought that was a slander,

because what actually happened was so easily proved to the sceptical: if a member would only attend the meetings of his society and join in the discussions his name and remarks would be most faithfully recorded and, in due course, would appear in the journal of the Association: if he would tactfully leave the journal in a readily-accessible spot, the truth would prevail and joy reign in the home. (Laughter.)

The dance which was to follow, and for the inception of which the President was undoubtedly largely responsible, was due to take place at once, therefore he would not detain them longer. November the fifth was an historical date, and he did not desire to lay himself open to the possibility of being held up as a guy. (Laughter.)

He did wish them to understand that he most fully appreciated the honour the Society had conferred upon him in awarding him the Victory Medal. (Cheers.)

After a short interval the company entered upon a most enjoyable dance programme (Professor G. H. Wooldridge officiating most acceptably and efficiently as M.C.), with interludes during which highly-appreciated items were rendered by Mr. Douglas Craggs "in ventriloquial humour and magic," and "Jacqueline" at the piano. Red Smith's Band gave the utmost enjoyment and satisfaction with their dinner selections and dance music.

## Victoria Veterinary Benevolent Fund.

A meeting of the Victoria Veterinary Benevolent Fund was held at 10, Red Lion Square, London, W.C.1, on Wednesday, October 7th, 1931, when there were present Lieut.-Colonel J. W. Brittlebank (President) in the chair, Professor J. F. Craig, Messrs. G. H. Locke, J. W. McIntosh, G. P. Male, Major-General Sir John Moore, Lieut.-Colonel P. J. Simpson, Captain Sidney Villar, Mr. W. Woods and Captain W. G. Wragg.

Apologies for absence were received from Major F. L. Gooch, Mr. J. Ewing Johnston, Captain J. R. Rider, Major C. W. Townsend and Mr. W. Jackson Young.

The minutes of the previous meeting, having been published in the *Veterinary Record*, were taken as read and signed as correct.

### Secretary's Report.

The Secretary reported that, since the previous Quarterly Meeting, the following new subscriptions and donations had been received:—

*Subscriptions.*

| | £ | s. | d. |
|---|---|---|---|
| Carless, W. J. ... ... ... ... ... | 1 | 1 | 0 |
| Duncan, Major A. C. (increase) ... ... | 0 | 10 | 6 |
| Eccles, A. ... ... ... ... ... ... | 0 | 10 | 0 |
| Hide, H. H. (increase) ... ... ... ... | 0 | 9 | 6 |
| King, H. C. P. (increase) ... ... ... | 0 | 10 | 6 |
| Knowles, Major R. H. ... ... ... ... | 1 | 1 | 0 |
| Male, G. P. (special) ... ... ... ... | 50 | 0 | 0 |
| Polding, J. B. ... ... ... ... ... | 0 | 10 | 0 |
| Pollard, H. (increase) ... ... ... ... | 0 | 10 | 0 |
| Sim, Mrs. F. ... ... ... ... ... | 5 | 0 | 0 |
| Tipper, L. C. (special) ... ... ... ... | 5 | 5 | 0 |
| Whicher, A. (special) ... ... ... ... | 1 | 0 | 0 |
| | £66 | 7 | 6 |

*Donations.*

| | £ | s. | d. | £ | s. | d. | |
|---|---|---|---|---|---|---|---|
| *Collecting Boxes*:— | | | | | | | |
| Baker, E. Whitley ... ... | 1 | 7 | 0 | | | | |
| Central Veterinary Society ... | 0 | 11 | 7 | | | | |
| | | | | 1 | 18 | 7 | |
| Dobson, N. ... ... ... ... | | | | 0 | 10 | 6 | |
| Eastern Counties V.M.S. ... ... | | | | 5 | 0 | 0 | |
| Fisher, Major R. C. U. ... ... ... | | | | 10 | 10 | 0 | (L.M.) |
| Heatley, Major T. G. ... ... ... | | | | 2 | 0 | 0 | |
| Masheter, J. W. H. ... ... ... | | | | 1 | 0 | 0 | |
| Richardson, Major J. M. ... ... | | | | 10 | 10 | 0 | (L.M.) |
| Royal (Dick) Veterinary College Alumnus Association ... ... | | | | 2 | 3 | 6 | |
| Smart, W. W. ... ... ... ... | | | | 1 | 1 | 0 | |
| Stothert, Major W. ... ... ... | | | | 1 | 1 | 0 | |
| Walker, Mrs. ... ... ... ... | | | | 4 | 4 | 0 | |
| Yorkshire V.M.S. (special) ... ... | | | | 5 | 10 | 3 | |
| | | | | £45 | 9 | 10 | |

It was resolved (*a*) That the grateful thanks of the Council be accorded to the respective donors, with special mention of Mr. G. P. Male for his very generous gift.

(*b*) That Major R. C. U. Fisher and Major J. M. Richardson be elected Life Members of the Fund.

### Cases.

No. 115. Member, unemployed.—The Secretary reported that he had given this member small sums from time to time, and his action was approved.

No. 133. Widow, aged 51, two sons.—A further application for assistance was received, the Council having discontinued their grant of £1. It was decided that, in the present state of the Fund, no further assistance could be granted.

No. 144. Daughter of member, aged 42, two children; 14 and seven.—An application was received for a grant of £2 17s. to enable this applicant to travel to and from hospital for special treatment. The application was acceded to.

No. 156. Widow, aged 46, two sons, aged 19 and 14; eldest earning £2 a week, younger on Training Ship.—The Secretary reported that during the quarter this applicant had been assisted to the extent of £14 14s. and that as she was seriously ill and totally unable to work further assistance was imperative. The matter was left in the hands of the Executive.

### Ladies' Guild.

The Hon. Secretary, Lieut.-Colonel P. J. Simpson, reported that already 30 ladies had promised to support the formation of a Ladies' Guild, and that the regulations had now been drafted. He hoped that by the beginning of the New Year he would be able to report that the Guild had commenced to function.

A Committee of the Empire Marketing Board has been appointed to consider a scheme to bring Empire frozen meat to this country in cartons. The Committee was appointed following a report by Dr. T. Moran, who has been carrying out new freezing methods at the low-temperature research station at Cambridge. In his report he says that though there may still be difficulties to be overcome before a trade can be developed, they are commercial, not scientific.

# Notes and News.

The Editor will be glad to receive items of professional interest for inclusion in these columns.

## Diary of Events.

Nov. 16th.—N.V.M.A. Editorial Committee Meeting at 2 Verulam Buildings, Gray's Inn, W.C.1.

Nov. 18th.—Meeting of Special Committee, N.V.M.A., at 2, Verulam Buildings, Gray's Inn, W.C.1, 3 p.m. Meeting of Finance and General Purposes Committee, N.V.M.A., at 2, Verulam Buildings, Gray's Inn, W.C.1, at 4 p.m.

Nov. 24th.—Annual General Meeting of the Dumfries and Galloway Division, N.V.M.A., at Dumfries.

Nov. 27th.—Meeting of the Royal Counties Division, N.V.M.A., at 10, Red Lion Square, London.

### Royal Veterinary College and the Lord Mayor's Show.

An Enlivening Element.

True to its purpose, nothing in the City of London's magnificent annual pageant stimulated the interest of the vast throng lining the route of the Lord Mayor's Show on Monday last, more than the strikingly effective little section representing the activities of the Royal Veterinary College. In addition, it was left to our students to provide the comic relief which is so essential and welcome a feature of all good shows, and of which the London public exhibited unstinted appreciation. Neither Sir Phené Neal and Sir Maurice Jenks (last years' Lord Mayor and his successor) have given more acceptable proof of their well-known interest in the veterinary profession than by seconding the efforts of Mr. Dixon Taylor (Secretary to the Lord Mayor and Sheriffs Committee) to enable the College to gratify its desire to bring its presence and its needs to the notice of the Londoner in this uniquely-effective manner.

As the Veterinary Section approached, the crowd (hitherto stolidly watching marching troops and the imposing parade of industry) brightened visibly and audibly, and stood tip-toe again to read the inscriptions on the College banner—" Help to make the Royal Veterinary College worthy of your City "; " Founded 1791: Falling Down 1932." The first car was a tableau representing modern veterinary science. Four Suffolk Punches drew the car, which was gaily decorated in the College colours (green and white) and the sides of which provided more thought-provoking material for the Londoner—" Tuberculosis can be controlled by Veterinary Science "; " We have saved your dogs from rabies "; " We have stamped out glanders from your horses," while, with quite unbelievable application, veterinary students addressed themselves to the laboratory apparatus impressively displayed on the vehicle. But the crowd was only momentarily hypnotised into impressed solemnity, for immediately behind followed the veterinary student in disguised but yet more recognisable form—giraffes, bears, monkeys and horses provoked their hilarity, as did the banner which announced " We treat camels, but we haven't got the hump," heralding " O'Treetemkind's Veterinary Hospital "—also drawn by Suffolk Punches. On this remarkable " bed " reclined student " patients " with animals' heads [Professor —— (from the crowd) " Realistic touch that "] dealing out dope from mammoth bottles and pills of tennis-ball proportions.

An amusing feature was an encounter with King's College students and their mascot which befel the R.V.C. contingent on the Embankment, where encouragement and loud cheering of the " foe " must have mystified the red lion completely.

The arrangement of the R.V.C. section was in the capable hands of Mr. Raymond Holmes, a Class C student, who thus adds another to the notable triumph he achieved in organising the College Carnival in North London and which raised over £1,000 towards the funds of the College.

### R.C.V.S. Obituary.

Hughes, Thomas D., Woodstock, Oxon. Graduated Edinburgh, December 21st, 1898. Died November 2nd, 1931; aged 64 years.

. . . . .

Jagger, Lieut.-Colonel Hugh Cleivion, O.B.E. Graduated Edinburgh, December 19th, 1894. Died November 6th, 1931, age 59 years.

. . . . .

The Late Mr. T. D. Hughes, M.R.C.V.S.

" The death, in his 65th year, of Mr. T. D. Hughes, of Oxford Street, Woodstock, has deprived the town of a well-known veterinary surgeon, and a man whose circle of friends extended over a wide area," says *The Oxford Mail*.

" Mr. Hughes, who had lived in Woodstock for 39 years, was more of a mutual friend and confidant than a veterinary surgeon to the farmers in the area, for he was ever to be seen mixing with farmers from all over the district, and his duties as secretary to the Woodstock Fat Stock Show brought him into contact with many more, who found that he was a capable business man.

" Mr. Hughes leaves a widow and a son and a daughter.

" His death came after an illness extending over six months, and it is understood that his heart was affected.

" During the latter part of his life a locum tenens had been carrying on his work, and the *Oxford Mail* understands Mr. Hughes had recently sold his practice.

" For many years Mr. Hughes was secretary of the Flower Show, and acted in a similar capacity to the Fat Stock Show until his illness kept him from attending to his work.

" He was W. Master of the Marlborough Lodge of Freemasons in 1908, and during the War his work for the Red Cross branch earned him many tributes."

. . . . .

The Late Lieut.-Colonel H. C. Jagger.

In the death of Lieut.-Colonel H. C. Jagger the Royal Counties Division of the N.V.M.A. has lost a valued member of over thirty years' standing, and who occupied the Presidential chair in 1905. The Society was represented at the interment, which took place at Woodstock on Tuesday last, by the Hon. Secretary (Mr. H. Bell, of Maidenhead) and Captain H. B. Collet, of Aylesbury, while the great number of floral tributes included one from the members. The gathering at Radclive Church was very large and representative, for Lieut.-Colonel Jagger, who served in the Boer War and the Great War, had many military friends, while the fact that his practice included a wide hunting connection added materially to the number of those present.

The deepest sympathy of the very large number of veterinarians who were privileged to know Lieut.-Colonel Jagger will be extended to the widow and two sons in their great loss.

### Personal.

*Illness of Captain Wyley.*—His numerous friends will regret to learn that Captain E. H. Wyley, M.R.C.V.S., of Henfield, Sussex, is laid aside with serious pulmonary trouble. It is hoped that this very popular and energetic member of the veterinary profession in West Sussex may soon be restored to his usual good health.

. . . . .

*Mr. A. Levie's Local Election Success.*—Mr. A. Levie, F.R.C.V.S., County Veterinary Officer, Derby, was successful by over 1,000 votes (in a poll of 4,000) in wresting the Rowditch Ward from its Labour representative at the recent Derby Municipal Election—a factor in a Conservative victory which ends Labour rule in the Derby Council.

### Army Veterinary Service.

London Gazette—War Office—Regular Army.

November 6th.—Major R. E. Bond is restored to the establishment. (November 5th).

Movements of Officers.

Major J. Mullany was posted from the Northern to the Eastern Command for duty, and joined at Woolwich on October 17th.

Major J. J. M. Soutar, o.b.e., sailed for India on October 30th, for a full tour of duty abroad.

Major R. E. Bond was restored to the British establishment from the Indian Continuous Service Cadre on November 5th, and has been posted to Aldershot for duty.

Major W. M. Rouse sailed for Egypt, and Lieut. R. K. Kent for India, on November 7th, for a full tour of duty abroad.

# Correspondence.

**Letters to the Editor should reach the Office not later than by the first post on Tuesday morning for insertion in following Saturday's issue.**

**All correspondence must bear the name and address of the contributor for publication.**

**The Editor does not hold himself responsible for the opinions of his correspondents.**

### Tuberculosis in Swine.

To the Editor of The Veterinary Record.

Sir,—In reply to Mr. Anthony's letter concerning my paper on "Tuberculosis in Swine," reported in the *Veterinary Record* of November 7th, I wish to state that I have no doubt that it may be possible for Mr. Anthony to find tiny lesions of tuberculosis in Free State pork heads, especially under the minute examination which he has found it practical to apply—the specially constructed light, I am sure, adds to the efficiency of such examination. However, it is my duty in this centre to supervise the inspection of Veterinary Meat Examiners, and I have frequently checked their work. Like Mr. Anthony, I too have found a small lesion in a submaxillary gland, but they are so few in number that they cannot materially affect my figures. I believe that the discrepancies in the figures quoted by Mr. Anthony and myself are due to the different systems of inspection in the Irish Free State Centres and in Messrs. Marsh and Baxter's Factory. As regards our system of inspection at this centre I would like to mention that during the slaughter of pigs there are two Veterinary Examiners in attendance—one who examines the heads while the other Examiner makes a detailed examination of the offal, examining as a matter of routine in all cases the chain of mesenteric glands, the bronchials and the portals. It is, I believe, this latter detailed examination which is responsible for the discrepancies between Mr. Anthony's figures and mine.

I would finally like to add that in this centre here, as well as several other large centres in the Free State, the general system of inspection in existence can scarcely be excelled.

Yours faithfully,
John J. Dundon,
*Chief Veterinary Officer.*

Veterinary Department,
Athenæum Buildings, Limerick.
*November 9th*, 1931.

### Sterility Research.

To the Editor of The Veterinary Record.

Sir,—With further reference to the letter in the *Veterinary Record*, Vol. xi, No. 44, October 31st, 1931, under the above heading, arrangements have been made whereby an examination of such specimens as are forwarded by practitioners will be carried out in connection with a survey of bovine sterility which is being undertaken by the aid of facilities provided by the **Scottish Sterility Research Committee** at the Royal (Dick) Veterinary College. It will save time, therefore, and considerably help matters if specimens, together with all information and communication relating thereto, are sent direct to Professor W. M. Mitchell at the Royal (Dick) Veterinary College, Edinburgh, and not to this Institute.

I am, Sir,
Yours faithfully,
W. C. Miller.

Institute of Animal Genetics,
King's Buildings,
West Mains Road,
Edinburgh.
*November 4th*, 1931.

### "The Eradication of Johne's Disease."

To the Editor of The Veterinary Record.

Sir,—Will you allow me to correct one or two glaring and obvious misstatements which are present in the published account of an article appearing under my name in your current issue of the *Veterinary Record?*

Owing to an administrative error, for which I accept full responsibility, the copy forwarded for publication was, most unfortunately, an uncorrected first draft.

The sentence appearing on p. 1093 col. 1 reads: "But even if we have this in mind . . . . 30 to 40 years." This should read: "But even if . . . . that it has been recognised and well known . . . . 30 to 40 years."

The first sentence in para. 3 col. 1 of the same page reads as follows:—"If, therefore, we assume that somewhere about 1901 . . . ." This should read: "If, therefore, we assume that somewhere about 1801 . . . ."

These errors are so obvious and so glaring that I hasten to make the necessary corrections lest it should be thought that they represent a correct interpretation of my views.

The "diarrhœa and wasting" described by Farrow and Cartwright in the *Veterinarian* in 1831 probably referred to what is now known as Johne's disease. This was, I believe, the first authentic description of the disease in English literature but, as is well known, it was not until 1895 that Johne and Frothingham described the acid fast bacillus which they found in the typically altered intestinal wall.

I am,
Yours faithfully,
George W. Dunkin.

The Farm Laboratories,
Mill Hill,
N.W.7.
*November 2nd*, 1931.

### "Sterility in the Mare."

To the Editor of The Veterinary Record.

Sir,—In your issue of November 7th, among the criticisms on my paper on "Sterility in the Mare," given at the Norwich Congress, it is represented from figures I have used that I have stated that 25 per cent. of thoroughbred mares are suffering from metritis.

It will readily be understood that I cannot possibly allow this to pass uncontradicted. I emphatically deny that any figures I have ever employed could rightly be taken to mean any such thing. That it should have been attempted so to interpret them is the more surprising since I stressed a very definite warning in my paper against the committing of this error. I will quote my words: "It has been estimated that at least 30 per cent. of the thoroughbred mares bred in any one season prove to be barren. I would point out that this should not by any means be taken to infer that 30 per cent. of thoroughbred mares are chronically barren. I stress this point because it would appear, from some views that have from time to time been expressed, that this is an error too frequently made."

Further, Colonel Todd in his opening of the discussion drew particular attention to this very point.

I am,
Yours faithfully,
E. Brayley Reynolds.

Newmarket.
*November 10th*, 1931.